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THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—16TH YEAR. SYDNEY, SATURDAY, APRIL 20, 1929. No. 16.

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An Address.[1]

By J. E. V. Barling, M.B. (Sydney),
Retiring President, New South Wales Branch of the British Medical Association.

Before undertaking my final official duty and delivering the valedictory address, I have again to thank you for the honour conferred upon me by my election to the presidency of this Branch. The full meaning of the compliment is better understood at the completion of one's term of office than at its beginning.

The insight which, of necessity, one gets into the inner workings of the Branch during one's occupancy of the chair, has given me a much keener appreciation of the magnitude of the Branch's work in the past, whether scientific, ethical or medico-political, and has engendered an enthusiastic belief in the great part which the Branch is destined to take in the advancement of medical science in the future and the application of new knowledge to the prevention and cure of disease.

The annual report which is in your hands, testifies to the Branch's activities during the past year. That report shows that the year has been an unusually busy one, both in regard to scientific work and in matters connected with internal administration, so that the various committees, to which are entrusted the manifold workings of the Branch, have been kept fully employed.

It has been found from experience that the various branches of our work are handled more satisfactorily if entrusted to small committees which are responsible to the Council.

New Committees.

During the year it has been found advisable to create two new standing committees; one to be known as the Post-Graduate Work Committee, whose members will, in conjunction with the Organization and Science Committee, undertake the supervision of post-graduate work and place our activities in this direction on a permanent and systematized footing. The other new committee to be formed will be known as the Hospital Committee, whose duty as its name implies will be to consider and report to the Council upon questions as to hospitals and other medical charities and the professional interests of the medical officers of those institutions. In both cases the committees have power to coopt members of the Branch who are not members of the Council, but who may have special qualifications for dealing with any particular question, and are willing to put their services at the command of the profession.

The importance of the work with which these two bodies will have to deal, cannot be exaggerated. It is certain aspects of their work that I particularly want to consider tonight.

[1] Delivered at the annual meeting of the New South Wales Branch of the British Medical Association on March 21, 1929.

Post-graduate Work.

First, as to the post-graduate work of the Branch. Its activities in this direction have been somewhat spasmodic, since post-graduate courses were started about eight years ago. In the early days they were organized by the Council and conducted by the University Extension Board. Later, the Organization and Science Committee became responsible and some excellent courses have been provided. In the main, however, up to the present our efforts have been lacking in continuity, coordination and system. With a permanent committee of enthusiastic members who have had some experience of this class of work, and who are alive to the needs of the profession, these defects are, we hope, soon to be remedied. The university authorities, the staffs of teaching hospitals and research workers have in the past shown that they are willing and eager to help in this direction and their cooperation can be relied upon for the future. So that once the committee gets to work, we may hope to have regular refresher courses in medicine, surgery, obstetrics and diseases of children, with periodic practical courses and demonstrations in applied anatomy and physiology and bacteriology *et cetera*. It will be their duty, therefore, to watch the pioneering work of the medical investigators and research workers, to see that new ideas are brought under the notice of the profession, so that established methods may be improved, conservatism discouraged and the profession kept alive by fresh currents of criticism and suggestion. Ultimately, no doubt, when our hospital systems have been perfected, we shall have hospitals set apart for post-graduate and research work. In the meantime we are looking to the committee to fill the gap.

As being a very essential element in medical education I should like here to refer to the Branch's library and the plans which the Council has in mind for its development. In Australia the profession generally and those doing research work particularly are at a great disadvantage, in that we have nothing comparable to the great medical libraries of England and America. We have, however, a very creditable collection which, as funds become available, can be added to and in our new building ample provision is being made for library accommodation and extension. In the near future, then, we hope to be able to extend the library's activities and usefulness by adding to our standard works, increasing the list of current periodicals and establishing a lending branch for the benefit particularly of those in country districts.

Hospital Reform.

As regards the work of the Hospitals Committee, if the bill now before Parliament becomes law, this committee will be concerned not only in developing a hospital policy, but also in cooperating with the authorities charged with the duty of developing a hospital system which shall meet all the needs of the community and at the same time contribute

to the future development of medical science and the continued progress of the medical profession.

In this State the problem is particularly vital and pressing on account of recent legislation. Probably no more important measure has ever been introduced into our local Parliament than the recent Hospital Bill, for upon the wisdom or otherwise of its provisions will depend not only the standard of public health, but also the future progress of medicine and the profession. The words of Herophilos, philosopher and physician, are as true today as when uttered centuries ago: "That science and art have equally nothing to show, that strength is incapable of effort, wealth useless, eloquence powerless, if health be wanting."

In the interest then of public health, as well as for reasons of economy, governments throughout the world equally with the profession are demanding that medical activities should be coordinated and systematized, so that the whole of the resources of the healing art should be at the disposal of the community, both as regards curative and preventive medicine. Each year our State Government is extending its activities in the realm of preventive medicine and has established a school, medical services, baby health centres, ante-natal clinics, tuberculosis dispensaries and so forth and has made itself responsible for a very large proportion of the capital and current expenses of our general hospitals, as its share in the provision of the curative agencies. The demand is then for adequate hospital facilities and a much wider extension of principles of preventive medicine.

In seeking for a solution of this all-important question all preconceived notions and prejudices, whether private or professional, political or provincial, must be put aside. The idea of the common health must be paramount. If our newly appointed Hospital Committee approaches the question with this idea uppermost in any endeavour to find a solution of the problem, it will render a service of inestimable value to the community and the profession.

The Hospital Bill then contains the solution of the problem as the Government sees it. In the circumstances it may be of general interest to review briefly some aspects of the question which have been discussed so fully in the medical journals during the year. These discussions deal chiefly with the professional side of the question, a point of view which is of great importance and apt to receive insufficient attention.

For my present purpose the subjects discussed may be roughly divided into: (i) The importance of hospitals to the State, their function and future development, (ii) the relationship of private practice and general and special practitioners to hospitals, (iii) the provision of pay beds, and payment by patients for relief, (iv) regulation of out-patient work at hospitals.

(i) The importance of hospitals to the State has already been touched on.

As regards their functions and future development Mr. R. G. Hogarth[1] has pointed out that they can no longer be regarded as "a charitable refuge for the destitute and a home of compassion for the sick." Owing to the strides made in all departments of medicine, they have become centres of medical science, in which are concentrated the best medical and surgical skill and the best equipment. Consequently, their resources should be at the command of the whole community. He concludes: "Let us aim to make the general hospital of the future one in fact as well as name, where the indigent sick are treated free, where the less poor will contribute according to their poverty, the less well-to-do according to their necessities and the well-to-do according to their means."

This view of the case evidently corresponds with that held by the framers of the bill in setting up a hospital commission, amongst whose duties will be to coordinate the activities of public hospitals, to survey the whole hospital situation of the State and to assist the hospitals in the extension of their functions in the direction of the establishment of hospitals suitable to all classes of the community.

(ii) As regards the second aspect discussed, the relationship of private practice and general and special practitioners to hospitals, the view is universally held that it is to the interest both of the public and of the profession that all practitioners should participate much more widely in hospital systems than at present; that from the patient's point of view it is better for him to be treated by his family doctor inside the hospital as well as out; and from the professional point of view, more widely distributed opportunities for hospital practice must tend to raise the proficiency of all members of the profession and enable them to render more efficient service to the public.

The principle is laid down then that practitioners must be in future more closely associated with hospital practice on broad educational grounds and that ultimately no competent practitioner will be debarred from such practice. Insistence is laid on the fact that only under these conditions can private practice and a satisfactory hospital system coexist.

(iii) The question is discussed as how this ideal can be attained. The solution suggested is said to be found in providing intermediate beds and private wards in association with general hospitals, in separate wings or buildings, if necessary, but so placed that the whole of the resources of the hospital should be at the command of the patient and the practitioner, including pathological and biochemical laboratories, X ray plant and the assistance of other specialties. Here I would quote from an article by Dr. C. M. Wilson.[2] He says:

> The day is coming when these pay beds will be found in every institution, great or small, when every doctor in the land and not a mere fraction of the profession as now will have frequent opportunities of treating his cases under

hospital conditions, where his diagnosis must pass the test of half a dozen laboratories before it is established, where new knowledge automatically is put before him in process of proof and where the cold wind of criticism blows upon his credulity. He will remain in contact with the hospital not for a few years before graduation, but throughout his professional life, with incalculable gain to himself and to the whole practice of his calling. I say deliberately that a change of this kind coming quietly as a side issue to a new demand upon the hospitals, might, by raising the standards of professional efficiency, do more for the health and happiness of the people than many years of considered legislation.

Referring now to the Hospital Bill, what provisions do we find for dealing with the establishment of pay beds? In the first place, the commission may and at the request of any hospital shall inquire whether it is advisable that any portion of a hospital should be so set apart for the admission of patients able to pay for private or intermediate accommodation; and the commission may, after inquiry, authorize the setting apart of any such portion.

As bearing on the financial aspect of the question the board of a hospital may, if the commission so recommends and the Governor approves, issue bonds or debentures for specified objects, amongst others the establishment of pay beds. These bonds are backed by the Government.

The liability of patients able to pay is established and powers are given to hospital boards to recover the cost of treatment. Also the commission may make regulations for the collection of fees for the services of medical men, though no medical man shall charge a patient under treatment in a general hospital, except on conditions prescribed by the commission.

(iv) Turning now to the work of out-patient departments, that this side of the work needs coordination and systematization is evident. The commission will probably approach the subject on the lines laid down by Mr. J. R. Love, before the Victorian Branch in March last year. He then advocated the establishment of consulting clinics in mid-suburban areas. These centres would act as clearing houses and only those "physically and financially suitable" sent on for hospital treatment. Attached to these clinics or centres would be waiting and examination rooms for patients sent by private practitioners who desire an investigation and report on any special condition and, possibly, tuberculosis dispensaries, ante-natal clinics, baby welfare centres *et cetera*.

The brief summary which I have presented, is necessarily incomplete, but it will serve to emphasize some of the problems which await solution. They can only be satisfactorily solved for the public if all concerned, commissioners, hospital boards and committees and members of the profession approach the subject in the spirit of cooperation and with the determination to do all in their power to coordinate the medical services of the State, so that greater efficiency may be procured and the knowledge of preventive and curative medicine, already in our possession, placed at the disposal of the whole community.

This much may be said of the present bill, that it is a step towards coordinating the medical activities of the State under one authority, with a view to developing a comprehensive hospital system. There are difficulties in the way, but the issues are so vital that any honest attempt should have our whole-hearted support.

We recognize that the profession is a "functional society" organized for the performance of duties, not merely for the maintenance of rights, and that it is possessed of that spirit indicated by Bacon, when he said that the work of men ought to be carried on "for the glory of God, and the relief of man's estate."

References.

(1) R. G. Hogarth: "Paying Patients in Hospitals," *The British Medical Journal*, Supplement, December 15, 1928, page 257.
(2) C. M. Wilson: "Pay Beds and the Future of the Voluntary Hospitals," *The British Medical Journal*, Supplement, March 17, 1928, page 85.

OBSERVATIONS ON THE HYDROGEN ION CONCENTRATIONS AND FLORA OF INFANTS' STOOLS.

By Lorimer Dods, M.B., Ch.M. (Sydney),
Sydney.

The subject matter of this study consists of a report on the naked eye appearances, pH values and intestinal flora of more than two hundred specimens of meconium and fæces from infants of less than fourteen months of age.

The majority of the specimens was collected from infants in the wards of the Royal Alexandra Hospital for Children, from infants at the Tresillian Mothercraft School, from infants attending a baby health centre and from new born infants at the Royal Hospital for Women.

It is hoped that the evidence presented, although admittedly slender, may be regarded as affording some support to the few original suggestions which have been put forward in this paper.

Naked Eye Appearances of Stools.

The Normal Infant.

Text books usually describe the normal stool of a healthy breast-fed infant as orange or golden yellow in colour and of a uniform semi-solid consistency.

The great majority of the stools from healthy breast-fed infants was found to agree with this description. But of the fifty-eight specimens collected, definite deviations from the normal naked eye appearances were noted in ten. The pH values and flora of these specimens showed no obvious deviation from those of other nursling stools and the infants involved were apparently in perfect health, showing no evidence of any enteral or parenteral infection.

The stools of normal healthy infants fed on a fresh cow's milk formula were found to be of a less uniform and more solid consistency and a little paler in colour than those of the breast-fed infant. Distinct deviations from the normal appearances were noted in five out of twenty-three specimens; there was no corresponding alteration in the pH value or flora of these five specimens.

Specimens of fæces from eighteen healthy infants fed on a dried milk formula, although similar in most respects to those following feedings of a cow's milk formula, were usually much more paste-like, firmer in consistency and in the majority of cases paler in colour.

The majority of the infants fed on a lactic acid milk formula passed stools which appeared to be almost identical macroscopically with those of the healthy breast-fed infant. In a few instances these feedings resulted in semi-liquid yellow stools and in three the stools were hard, dry and pale.

Observations of specimens of fæces from fourteen breast-fed infants, suffering from various parenteral infections, showed that variations from the normal colour and consistency occurred in eleven specimens. These eleven specimens varied from stools of normal consistency, showing slight changes in colour, to frequent greenish-yellow or green, semi-liquid stools. The stools from three of these infants showed no deviation from the normal appearance.

It would seem that the nature of the stools from an apparently healthy infant depends on the infant's diet, his digestive powers, the rate of absorption from the bowel and the intestinal flora.

The first and the last factor have been considered in some detail, but it must be realized that slight changes in one or both of the remaining factors may cause changes in the appearance of the infant's stool without producing any obvious deviation from his normal health and vigour.

Acidity of Stools.

There are probably many factors which may influence, to a major or minor degree, the acidity of infants' stools. The great majority of these factors falls into one of the following groups:

1. The intestinal flora.
2. The diet.
3. Enteral or parenteral infections.

The Intestinal Flora.

The original theory, held by Metchnikoff and others, that the type of the predominating organism determines the acidity of the intestinal contents has recently been contradicted by several investigators, including Ylppo and Czerny and Keller. These observers advance the view that the acidity of the intestinal contents determines the type of the intestinal flora. This is apparently the generally accepted theory today.

The Diet.

The rather contradictory literature on this subject seems to suggest that the sugar content of an infant's diet is the chief factor in the production of stool acidity. But attempts to alter the reaction of the intestinal contents by feeding with various sugars have produced very variable and somewhat unsatisfactory results. Sisson fed young puppies on cow's milk formulæ containing high percentages of sucrose or lactose to the point of diarrhœa without producing any change.

Tisdall and Brown suggest that it is not so much the amount of sugar in the diet as the amount of sugar which remains unabsorbed in the intestine which is the important point in the production of an acid stool.

Bergeim states that the acidity of the stools of breast-fed infants is probably due to the relatively high content of lactose in human milk. In contradiction of this statement Norton and Shohl contend that when a milk mixture containing carbohydrate similar to that of breast milk is given to infants, the stools become alkaline.

Apparently the fat content of an infant's diet plays little or no part in increasing stool acidity, as fat utilization is almost complete (more than 90% of the fat intake being absorbed by the infant). Proteins produce no immediate changes in the acidity of the intestinal contents. Later changes may be produced by favouring the development of putrefactive organisms. It is doubtful whether variations in the base content of the diet play any part in modifying the reaction of the stools. Ylppo observed that in starvation a stool results with an average pH value of 6·4 and that the infant's previous diet has no effect on this figure.

Parenteral and Enteral Infections.

The variable changes produced in the acidity and flora of infants' fæces by these infections will be considered later.

The above statements would suggest that the acidity of the infant's stool varies according to the nature of the diet and the presence or absence of enteral or parenteral infection and that this acidity determines the type of intestinal flora.

Hydrogen Ion Estimations.

For an exact investigation of the acidity of a specimen of fæces, litmus paper tests are of little or no value and some method of estimating the hydrogen ion concentration of the specimen should be adopted.

The results obtained by testing with litmus paper are most inaccurate when compared with the estimated hydrogen ion concentration of the specimens tested. It was found that red litmus paper may fail to change colour when applied to specimens with alkaline pH values (even as high as pH 8·0) and that blue litmus paper may fail to change colour when applied to specimens with acid pH values (even as low as pH 5·0).

The few reported investigations of the hydrogen ion concentration of infants' fæces have been carried out by means of electrometric apparatus or by dialysis of the fæcal emulsions through collodion sacs, followed by the application of colorimetric tests to the dialysates.

The former method necessitates special apparatus and demands a technique which is possibly rather beyond the average observer.

The latter method (as described by Tisdall and Brown) is simpler, the difficulty experienced in obtaining a clear and colourless fæcal extract for colorimetric tests is overcome by dialysis; unfortunately this manœuvre makes the method a lengthy one.

The colorimetric method used in this study overcomes this difficulty of obtaining a clear and colourless fæcal extract, is simple, necessitates no special apparatus and requires comparatively little time. The results obtained by this method are admittedly less accurate than those obtained by the more complicated procedures, but should be sufficiently accurate to be of value to the clinician in a study of this subject.

Method.

The specimens were obtained by means of a short, straight piece of glass tubing, one end of which had been rounded. By inserting such a tube into an infant's rectum a small quantity of fæces was collected in its lumen.

This specimen of fæces was immediately dissolved in approximately four cubic centimetres of freshly distilled water. This method of collection insured a fresh specimen of fæces uncontaminated by urine or by contact with the infant's buttocks or diaper.

Estimations of hydrogen ion concentration were performed on all specimens within one hour of their collection. The technique used was a modification of that suggested by the British Drug Houses for use with their universal indicator.

Hydrogen Ion Concentration of the Stools of Normal Infants.

The possible relation of the acidity of infants' intestinal contents to growth, general health, dietetic errors, enteral and parenteral infections suggests that a study of the hydrogen ion concentration of infants' stools should be of value to the clinician.

An essential preliminary to such a study is the establishment of average fæcal hydrogen ion concentration for normal healthy infants fed on the more common food formulæ. With the object of establishing such values, the following estimations were carried out.

Meconium and the Stools of New Born Infants.

Specimens were collected from apparently normal healthy new born infants. Ten of these infants were inmates of the Royal Hospital for Women and five were infants seen in private practice. It was found that the majority of infants passed meconium for the first two days of life. Sometimes this persisted during the third and fourth days, but as a rule the typical nursling stool appeared on the third day. In some instances this nursling stool appeared even earlier than the third day. Probably this change depends on the ingestion of human milk and the onset of digestive processes.

The results of the estimations of hydrogen ion concentration have been tabulated in Table I and may be summarized as follows:

Average pH value on the first, second and third days of life .. .. 7·2

Average pH value on the seventh, eighth and ninth days of life .. 6·17

These figures demonstrate the change from an alkaline meconium to an acid fæces.

Table I.
The Stools of Healthy New-born Infants.

Day of Life.	Nature of Stool.	pH Value of Stool.	Stool Flora.
1st	Meconium	7·5	No organisms seen.
1st	Meconium	7·5	Scanty organisms; small Gram-negative bacilli, Gram-positive cocci.
2nd	Meconium	7·5	A few small Gram-positive bacilli, cocci (Gram-positive and Gram-negative).
3rd	Nursling	6·5	Many Gram-negative bacilli, numerous Gram-positive cocci.
4th	Nursling	5·0	Large Gram-negative bacilli, many Gram-negative cocci.
4th	Nursling	6·0	Gram-positive and Gram-negative bacilli, Gram-positive cocci.
5th	Nursling	7·0	Gram-negative bacilli and cocci.
6th	Nursling	6·5	Gram-positive bacilli (large).
6th	Nursling	5·5	Gram-positive bacilli (large), small Gram-negative bacilli, cocci (Gram-positive and Gram-negative).
7th	Nursling	7·0	Gram-positive bacilli (large), Gram-negative cocci.
7th	Nursling	5·5	Gram-positive and Gram-negative bacilli, Gram-negative cocci.
7th	Nursling	6·5	Gram-positive bacilli (large and small), some Gram-negative bacilli.
8th	Nursling	6·5	Numerous large Gram-positive bacilli, some small Gram-negative bacilli.
9th	Nursling	5·5	Large Gram-positive bacilli.
9th	Nursling	6·0	Large Gram-positive bacilli, Gram-negative bacilli and cocci.

In all these tables the word "cocci" includes diplococci, streptococci and ordinary single cocci. No note has been made of spores or yeast cells.

Breast-fed Infants.

The specimens from breast-fed infants were collected from vigorous, well nourished, healthy infants under eight months of age and have been divided into two groups: (i) Specimens from infants of two weeks to three months of age, (ii) specimens from infants of over three months and under eight months of age.

Group I. A total of eighteen specimens was examined. With two exceptions the estimated pH values varied between 4·5 and 6·0. The average pH value was found to be approximately 5·5.

Group II. A total of forty specimens was examined. Thirty-six of the pH values varied between 4·0 and 7·0. The average pH value was found to be approximately 6·0.

The average pH value of the stools of fifty-eight normal breast-fed infants under eight months of age was thus estimated to lie between 5·5 and 6·0.

Infants Fed on a Cow's Milk Formula.

The hydrogen ion concentration of the stools of twenty-three infants fed on a cow's milk formula was estimated. These infants were apparently normal and healthy and were between three months and eight months of age. Each infant had been fed on a cow's milk formula for at least two weeks before the collection of a specimen. This formula

comprises cow's milk, water, sugar of milk and cod liver oil emulsion and yields approximately 6·5% to 7·5% of carbohydrate, 3·2% to 3·5% of fat and 1·25% to 2·0% of protein.

The pH values obtained varied between 6·0 and 8·5; nineteen (that is approximately 82%) of the specimens had pH values of 7·0 to 8·0. The average pH value was approximately 7·5.

TABLE II.

Stools of Apparently Normal Infants Fed on a Cow's Milk Formula (Aged Three to Nine Months).

Age of Infant, Months.	Naked Eye Appearance of Stool.	pH Value of Stool.	Predominance of Gram-positive or Gram-negative Flora.	Gram-positive Bacilli (Large and Small).	Gram-negative Bacilli (Large and Small).	Gram-positive Cocci.	Gram-negative Cocci.
3	Pale yellow, normal	7·5	Neg.		++	p.	p.
5	Yellow, normal	8·5	Neg.	p.	++	p.	
8	Yellow, normal	7·5	Pos.	++	+	+	
5	Greenish-yellow, relaxed	7·0	Neg.		+++	p.	
6	Yellow, normal	7·5	Neg.		+++		+
3	Yellow, normal	8·0	Pos.	++	p.	p.	p.
4	Greenish-yellow, relaxed	7·5	Neg.	p.	++	+	
6	Pale yellow, normal	6·0	Neg.		+++	p.	
7	Yellow, normal	7·5	Neg.		+++		
8	Yellow, normal	8·0	Neg.		+++	p	p.
8	Pale and paste-like	7·5	Neg.	p.	+++		
5	Pale yellow, firm	7·25	Pos.	++	+	+	
4	Yellow, normal	8·0	Neg.		+++		
5	Yellow, relaxed	8·5	Neg.		+++	p.	
5	Yellow, paste-like	7·0	Neg.	p.	+++		p.
6	Yellow, normal	7·75	Neg.		+++	p.	
3	Yellow, normal	8·0	Neg.	p.	+++		
5	Green, semi-liquid	7·25	Neg.	+	++	p.	p.
6	Yellow, normal	8·5	Pos.	p.	++	++	
4	Yellow, relaxed	6·5	Neg.	+	++		
7	Pale yellow, paste-like	7·0	Neg.		++	+	p.
6	Clay-like, firm	7·5	Neg.		+++	p.	p.
4	Yellow, normal	7·5	Pos.	++	+	++	+

p. has been used in these tables to denote that the organisms concerned were observed in the film, but were very scanty.

Infants Fed on a Dried Milk Formula.

The specimens were taken from eighteen healthy infants between two and eight months of age, every infant had been fed on a dried milk formula for at least two weeks before the collection of a specimen. It comprised dried milk, water, sugar of milk and cod liver oil emulsion and yielded approximately 7·0% to 7·5% of carbohydrate, 3·0% to 3·5% of fat and 1·0% to 2·0% of protein.

Of the eighteen specimens examined fifteen (that is approximately 82%) were distinctly alkaline (pH 7·5 to 8·5). Two specimens had an acid pH of 6·0. The average pH value was approximately 7·7.

TABLE III.

Stools of Apparently Healthy Infants Fed on a Dried Milk Formula.

Age of Infant, Months.	Naked Eye Appearance of Stool.	pH Value of Stool.	Predominance of Gram-positive or Gram-negative Flora.	Gram-positive Bacilli (Large and Small).	Gram-negative Bacilli (Large and Small).	Gram-positive Cocci.	Gram-negative Cocci.
7	Yellowish-white, semi-formed	8·0	Neg.	p.	++	p.	+
4	Greenish-yellow, semi-liquid	8·0	Neg.		+++		p.
4	Normal consistency, paler than usual	7·5	Pos.	++	+	+	p.
2	Pale yellow	7·75	?	++	++		p.
8	Yellow, normal	8·0	Neg.		+++		
3	Pale yellow, normal	8·5	Neg.	+	++	p.	p.
6	Normal	7·5	Neg.		+++	p.	+
4	Normal	7·0	Neg.	p.	+++		
7	Almost white, paste-like	7·25	Neg.		++	p.	p.
5	Normal	7·5	?	++	++	p.	
3	Pale and chalk-like	8·5					
8	Pale yellow, formed	6·5					
1½	Normal	7·0					
4	Normal	7·5					
6	Greenish-yellow, semi-formed	6·0					
2	Pale yellow, paste-like	8·5					
5	Normal	8·5					

Parenteral and Enteral Infections.

The parenteral and enteral infections will be considered when the effect of such infections on the fæcal flora is discussed.

THE INTESTINAL FLORA.

Several elaborate studies of the intestinal bacteria of the infant have been recorded, but there seems to be very little known concerning their modes of action, their significance and their relation to their host. Tissier, in describing the development of a normal intestinal flora in breast-fed infants, recognizes four stages which may be summarized as follows:

Stage I.—The meconium in the new born infant is sterile and may remain so for four to twenty-four hours after birth, after this period it is invaded by organisms from the infant's surroundings.

Stage II.—This invasion leads to a mixed infection by many and varied bacteria introduced into the infant's nose, mouth and anus. The immediate consequence is putrefaction of the meconium.

Stage III.—Following the ingestion of breast milk, a characteristic suckling flora is established. *Bacillus bifidus* becomes dominant and ousts the ordinary putrefactive bacteria.

Stage IV.—The introduction of artificial food disturbs the above order and the putrefactive species flourish.

Various investigators have shown that in the normal individual this bacterial growth is most prolific in the ileum and large intestine, there being relatively few bacteria in the jejunum and duodenum.

Whether these bacteria are or are not essential for the normal life of their host is still a disputed question, but they are found to be universally present and it is probable that they play an important part in the various intestinal processes.

The modern view that the acidity of the intestinal contents controls the intestinal flora and not *vice versa* has already been mentioned and is confirmed by the investigations of Ylppo and Czerny, Scheer and others.

Scheer inoculated various milk media with stools from infants fed on breast milk and infants fed on protein milk. The stools from the infants fed on breast milk were acid in reaction and the stools from the infants fed on protein milk were alkaline in reaction. In three days the acidity of all the media had reached a pH value of 3·7, regardless of the type of the stool with which they had been inoculated. Evidently acid-producing organisms were present in both types of stool, but apparently in the case of the stool from the infant fed on protein milk, the conditions were not favourable for their proliferation.

Ylppo has also demonstrated the very interesting fact that after fasting infants fed on either a breast milk or a cow's milk diet pass stools of approximately the same hydrogen ion concentration, an acid pH of 6·4.

Assuming, then, that the intestinal bacteria are controlled by the acidity of the intestinal contents and that they depend to a large degree on the food of their host for their sustenance, it would seem that it should be possible by certain dietetic measures to change the intestinal flora. Experiments have proved that this is possible, but tedious, difficult and somewhat unreliable.

In the case of the normal breast-fed infant, Nature appears to have provided especially for the maintenance of a characteristic nursling flora, the essential for the maintenance of this flora being breast milk. As soon as artificial feeding is substituted in part or in whole for breast feeding, this nursling flora is upset, another flora is established and, as will be suggested later, the infant becomes prone to gastro-enteritis and infections with organisms of the *Bacillus dysenteriæ* type.

If breast milk is the natural essential for the maintenance of the nursling's fermentative intestinal flora, is it possible that breast milk feeding would be of any therapeutic value in the acute intestinal disorders of the artificially fed infant as a means of restoring the fermentative flora?

With this question in mind, the stools of previously breast-fed infants who had been artificially fed for short periods and were returning to breast feeding, were examined. It was found that the intestinal flora of the artificially fed infant persisted for two or three weeks or longer after breast feeding had been fully reestablished.

M.K., a healthy, vigorous male, weighed 3·4 kilograms (seven and a half pounds) at birth. He was breast fed until two months of age when "vomiting and constipation" led to the substitution of "Lactogen" feedings for two and a half weeks. Breast feedings were then reinstituted and were complemented with modified "Lactogen" while necessary. On October 2, 1927, he was fed on modified "Lactogen" diet. The stools were pale yellow and paste-like and the pH value was 7·5; there was a distinct preponderance of Gram-negative organisms. On October 5 the diet was changed to breast milk and modified "Lactogen." The stools had the same appearance, but the pH value had been reduced to 8·0. There was no change in the organisms. On October 25 the diet was still the same. The stools were pale yellow and semi-solid and the pH value was 7·5. There was a predominance of Gram-negative organisms, but some Gram-positive rods were present. On October 29 the infant was being fed on breast milk only. No change in pH value was noted, there was a slight increase in the number of Gram-positive rods. On November 15, 1927, the diet was the same. The stools were typical nursling stools with a pH of 6·5. While Gram-negative organisms predominated, there were many Gram-positive rods.

William T. was breast fed every three hours until he was two and a half months of age, when as the result of neighbourly advice, four of the breast feedings were replaced by modified cow's milk feedings. When the infant was first seen it had been fed on a modified cow's milk formula for one month and had received breast milk each night and morning. On May 12 while it was having modified cow's milk and two breast feedings, it was passing greenish-yellow, semi-formed stools with a pH value of 8·0. There was a distinct predominance of Gram-negative organisms. On May 26 five breast feeds were being given with complementary modified cow's milk. The stools were pale yellow and semi-formed and the pH value was 8·0. No change in the flora was noted. On July 26 the infant had been fed exclusively on the breast milk for one month. The stools were pale yellow with a pH value of 8·0 and Gram-negative organisms still predominated.

E.V., an underweight female infant, had been breast fed irregularly until she was three months of age. Test feedings revealed that the supply was insufficient. It was therefore complemented with dried milk. Full breast feeding was reestablished in four weeks. On September 7, 1927, while on breast milk only, the stools were liquid and yellow with a pH value fo 6·0. There were numerous Gram-positive bacilli and a few Gram-negative bacilli. On September 8 dried milk was added. On September 19 the stools were green and semi-liquid with a pH value of 7·5. No change in the flora was detected. On October 25 breast milk had been given alone for four days. The stools were yellow and semi-formed. The pH value was 8·0. They contained Gram-positive bacilli and some Gram-negative bacilli. There were in addition some Gram-positive cocci. Three weeks after the breast feeding had been reinstituted the conditions were the same and the pH value was 7·5.

This slow transformation of the intestinal flora in infants who had been artificially fed for only short periods, would help to exclude breast milk feedings as a therapeutic measure in the acute intestinal disorders of artificially fed infants.

The question of reproducing a nursling flora in artificially fed infants by feeding cultures of *Bacillus lactis acidi* or a diet of lactic acid milk was also studied and will be considered later.

The following study of the intestinal flora of infants was limited to an examination of direct smears taken from the specimens collected for estimations and stained by Gram's method.

Brown and Courtney and Davis as a result of their investigations of the persistent infantile flora associated with chronic intestinal indigestion, state that "the examination of the stool smear was of the greatest importance." Lucas points out in his text book that "culturally we obtain a very inaccurate picture of the intestinal flora of fæcal specimens, as many of the forms present in the fæces are not viable on culture." Sisson, as a result of his studies of the intestinal flora, concludes that one cannot distinguish characteristic local flora in the intestines.

The above authorities have been quoted here in defence of the stool smears as opposed to more elaborate studies, as a method of reporting on the intestinal flora.

As this study of stool flora is purposely restricted to a microscopical examination of Gram-stained smears, no attempt will be made to distinguish bacteria, as *Bacillus bifidus, Bacillus welchii, Bacillus lactis aerogenes, Bacillus coli, Bacillus acidophilus* and many others. The only attempted classification will be a simple and primitive one according to size, form and staining reaction.

The Flora of Meconium and of the Stools of New Born Infants.

The specimens were collected from apparently healthy infants between eighteen hours and nine days of age. It was found that few organisms (and in one case no organisms) were seen in the smears from infants of less than two days and that the smears of infants between two and three days of age contained numerous Gram-positive and Gram-negative bacillary and coccal forms. Smears from infants between four and nine days of age showed a slight diminution in the number of cocci and a great increase in the bacillary forms (see Table I).

The Flora of the Stools of Healthy Breast-fed Infants.

Smears taken from infants under three months of age showed a predominance of Gram-positive organisms in 77%; those from infants over three months of age showed a predominance of Gram-positive organisms in 71%.

In the majority of the smears the predominant organism was a large Gram-positive bacillus (sometimes long and slightly curved, sometimes short and thick) which corresponded in appearance to the text book description of *Bacillus bifidus*. In what might be regarded as the typical smear of a breast-fed infant's stool, the field seemed to be almost completely obscured by a large number of these bacilli, cocci and Gram-negative bacilli being absent or very scarce.

Gram-negative bacilli were seen in many films and in a few cases were predominant. Some of these Gram-negative bacilli were similar in size and form to *Bacillus coli* and others were similar in size and form to *Bacillus bifidus*. In several films some of these large Gram-negative bacilli were noticed to contain one or two Gram-positive granules (see Tables IV and V).

TABLE IV.

The Stools of Apparently Normal Breast-fed Infants (Aged Two Weeks to Three Months).

Age of Infant.	Naked Eye Appearance of Stool.	pH Value of Stool.	Predominance of Gram-positive or Gram-negative Flora.	Gram-positive Bacilli (Large and Small).	Gram-negative Bacilli (Large and Small).	Gram-positive Cocci.	Gram-negative Cocci.
1 month	Normal	6·0	Pos.	+++	p		
2 weeks	Normal colour, but semi-fluid	5·5	Pos.	+++	p.		
3 months	Normal	5·5	Neg.		+++	p.	
3 months	Normal	5·5	Pos.	+++	+		
2 months	Normal	5·0	Pos.	+++		p.	
3 weeks	Normal	5·25	Pos.	+++	p.	p.	
1 month	Normal	4·75	Pos.	+++			
3 months	Normal	6·0	Neg.	+	++	p.	p.
6 weeks	Pale greenish-yellow, semi-liquid	5·5	Pos.	++	+	p.	
2 weeks	Normal	5·0	Pos.	++	+		p.
12 days	Normal	5·0	Pos.	+++	p.		
2 months	Normal	5·5	Neg.	p.	+++	p.	p.
2 months	Normal	7·0	Pos.	+	p.	+	
3 months	Normal	5·0	Pos.	+++	p.	p.	
1 month	Very pale yellow, otherwise normal	4·5	Pos.	+++			
6 weeks	Normal	6·0	Pos.	+++	+		
3 weeks	Greenish colour, liquid	5·75	Pos.	+++	p.	p.	
2 months	Normal	7·5	Neg.	p.	+++		

The Flora of the Stools of Healthy Infants Fed on a Cow's Milk Formula.

The flora of the stools of healthy infants fed on cow's milk formula was less homogeneous than that of the nursling. Gram-negative organisms (chiefly bacillary forms) predominated in 78% of the specimens examined, Gram-positive bacilli were scanty in number and in many cases apparently absent from the smear and coccal forms were more numerous (see Table II).

The Flora of the Stools of Healthy Infants Fed on a Dried Milk Formula.

Seven out of the ten specimens examined showed a predominance of Gram-negative bacillary forms. Gram-positive bacilli, Gram-positive and Gram-negative cocci were also present (see Table III).

The Flora and the Hydrogen Ion Concentration of the Stools of Breast-fed Infants Suffering from Parenteral Infection.

The predominance of Gram-positive bacilli, so characteristic of the stool of the normal nursling, was noted only in three out of twelve stools examined and of the remaining cases there was a

Table V.

Stools of Apparently Normal Breast-fed Infants (Aged Three to Seven Months).

Age of Infant, Months.	Naked Eye Appearance of Stool.	pH Value of Stool.	Predominance of Gram-positive or Gram-negative Flora.	Gram-positive Bacilli (Large and Small).	Gram-negative Bacilli (Large and Small).	Gram-positive Cocci.	Gram-negative Cocci.
7	Normal	6·0	?	+	+	+	
4	Normal	5·5	Pos.	++	+	+	
5	Normal	6·5	Pos.	+++	p.		
5	Normal	6·0	Neg.		+++	p.	
7	Brown, formed	7·5	Pos.	+++	p.		
5	Normal	5·5	Pos.	+++			
4	Normal	5·0	Pos.	+++	p.		
6	Normal	7·0	?	+	+		p.
7	Normal	6·0	Pos.	++	+		p.
4	Greenish-yellow, liquid	6·0	Pos.	+++	p.	p.	
4	Normal	5·5	Pos.	+++			
3½	Normal	6·0	Pos.	+++	+		
5	Normal	6·0	Neg.		+++		
4	Normal	5·0	Pos.	++	+		
3½	Normal	5·5	Pos.	+++			
7	Normal	7·0	Pos.	++	+	p.	
5	Normal	6·0	Pos.	+++	p.		
4	Normal	6·5	Pos.	+++			
5	Normal	5·5	Pos.	+++			
3	Greenish-yellow, semi-liquid	6·0	Neg.		+++		
5	Normal	6·5	Neg.		+++	p.	p.
7	Normal	7·5	Neg.	p.	++	p.	
6	Pale yellow, liquid	6·5	Pos.	++	p.		
6	Normal	6·25	Neg.		+++		
4	Normal	5·0	Pos.	+++			
5	Normal	5·0	Pos.	+++	+		
4	Normal	4·0	Pos.	+++	p.	p.	
7	Green, liquid	5·0	Pos.	+++			
4	Pale yellow, paste-like	5·75	Pos.	++	+		
4	Normal	5·0					
3	Normal	5·75					
5	Normal	5·0					
5	Normal	6·0					
7	Greenish-yellow, relaxed	5·5					
6	Normal	8·0					
6	Normal	6·0					
7	Dark yellow, liquid	5·25					
5	Normal	6·5					
3	Normal	6·0					
7½	Normal	6·0					

definite predominance of Gram-negative bacilli in five (that is approximately 42%).

Of the twelve specimens, eight (that is approximately 67%) had an alkaline pH value. The average pH value of the twelve specimens was 7·4 (see Table VI).

The Flora and the Hydrogen Ion Concentration of the Stools of Infants Suffering from Gastro-Enteritis.

The stools of twenty-eight infants suffering from acute gastro-enteritis, were examined. No pathogenic organisms were isolated from these stools by the usual cultural methods. The average pH value was found to be 7·9, the flora was a mixed one and Gram-negative forms were apparently predominant.

No obvious differences from these findings were noted in the hydrogen ion concentration or the flora of the stools of five infants suffering from a *Bacillus dysenteriæ* Flexner infection (see Tables VII and VIII).

Table VI.

Breast-fed Infants Suffering from Parenteral Infections.

Age, Months.	Remarks.	Naked Eye Appearance of Stools.	pH Value of Stool.	Predominance of Gram-positive or Gram-negative Bacteria.	Gram-positive Bacilli (Large and Small).	Gram-negative Bacilli (Large and Small).	Gram-positive Cocci.	Gram-negative Cocci.
1½	Severe pertussis of one week's duration	Pale yellow, liquid	7·5	?	+	++	+	
2	Acute *otitis media*	Frequent, green, semi-liquid	5·5	?	++	++	p.	p.
1	Pertussis of moderate severity for two weeks	Greenish-white, pasty, small curds	8·5	Neg.	p.	+++		
6	Mild degree of pertussis for five weeks	Greenish-yellow, semi-formed	7·5	Pos.	++	+		p.
3	Severe pertussis for ten days	Normal	8·0	Neg.		+++	p.	p.
4	Acute bronchitis (four days' duration)	Normal	5·5	?	++	++		
2	Broncho-pneumonia (ten days' duration)	Pale yellow, liquid	8·5	Neg.	p.	+++		
3	Pneumonia (six days' duration)	Greenish-yellow	6·5	Pos.	++		p.	p.
10	Pneumonia (six days' duration)	Very pale and paste-like	8·0	?	p.	++		p.
7	Pneumonia (four days' duration)	Watery, greenish-yellow	6·0	Neg.		+++	p.	
2	Acute bronchitis (? seven days' duration)	Normal	8·5	Neg.		+++	p.	
6	Measles (three days' duration)	Dark yellow, semi-liquid	8·0	Pos.	+++		p.	p.

During the summers of 1924-1925 and 1925-1926 605 infants were admitted to the Children's Hospital suffering from gastro-enteritis. A study of the hospital records shows that approximately 2·5% were totally breast fed, approximately 97·5% were totally or partially artificially fed, approximately 69·2% were between three and twelve months of age. The mortality among the breast-fed infants was approximately 8%; the mortality among the artificially fed infants was approximately 25%.

Of the fifteen breast-fed infants who suffered from gastro-enteritis, there was a history of a severe coincident parenteral infection in twelve infants.

Dysentery bacilli were isolated from the stools in only 17% of the patients admitted.

These figures help to illustrate the following points:

(i) That breast feeding apparently affords a strong protection against gastro-enteritis.

(ii) That the introduction of the commoner artificial feedings seems to make the infant prone to gastro-enteritis. In this connexion it is interesting to note that nearly 70% of the infections

TABLE VII.

Stools of Infants Suffering from Gastro-enteritis.

(No pathogenic organisms isolated from stool cultures. Specimens collected during acute stages, within three days of admission).[1]

Naked Eye Characteristics of Stools.	pH Value.	Gram-positive Bacilli.	Gram-negative Bacilli.	Gram-positive Cocci.	Gram-negative Cocci.
Greenish-yellow, mucus	8·5	p.	++		+
Greenish, semi-fluid, mucus	8·0		+++	p.	p.
Dark green, curds ..	7·5	+	+	p.	
Green and yellow, mucus and curds	8·5		+++		p.
Greenish-yellow.. ..	8·5		+++	+	+
Green, fluid, mucus and bloodin large quantities	8·0		++	p.	
Small dark green semi-formed stools	8·75		++	++	
Dark yellow liquid and curds	7·0	p.	+	+	p.
Green, relaxed, mucus and curds	7·5	++	+	p.	p.
Green, fluid	6·5		++	+	
Green, semi-fluid, mucus and blood	8·5		+	+	++
Small green stools with much mucus	8·5		+++		p.
Yellowish-green, semi-formed	7·75	+	p.	+	++
Brown, semi-liquid, much mucus	7·5	++	+		p.
Green and yellow liquid	5·75		++	++	+
Dark green, large curds	8·5		+++		
Green liquid, much blood and mucus	8·0	p.	++		+
Yellow, semi-liquid ..	6·5		+	+	+
Dark green, curds and mucus	7·5		+		+++
Greenish-yellow liquid..	7·5	+	+++	p.	p.
Green, semi-formed, mucus	7·5		+	+	++
Small green, semi-fluid stools with very much blood and mucus	8·5		++	++	p.
Green and yellow liquid	8·0	++	+		+
Yellowish-green, semi-liquid	6·0		++	p.	
Green, semi-fluid, mucus and blood	8·75		+	+	++
Greenish-yellow, streaks of blood and mucus	8·0		++	++	

[1] These infants were receiving nothing but whey and boiled water at the time the specimens were collected.

TABLE VIII.

The Stools of Infants Apparently Suffering from an Infection with Bacillus dysenteriæ (Flexner).

Naked Eye Appearance of Stool.	pH Value.	Gram-positive Bacilli (Large and Small).	Gram-negative Bacilli (Large and Small).	Gram-positive Cocci.	Gram-negative Cocci.
Motion consisted chiefly of blood and mucus, some green fæces	8·0	p.	++	+	p.
Small semi-liquid, greenish-yellow stools, much mucus and a little blood	8·0	+	++		+
Green liquid stool, blood and mucus present	7·0		+++		++
Palegreenish-yellowstool, much blood and mucus present	8·5	+	++	+	p.
Green semi-liquid stool, mucusandbloodpresent	7·5		++	p.	p.

occurred in children of three to twelve months of age, that is the period during which artificial foods are being introduced and breast feedings ceased or diminished.

(iii) That the mortality rate from gastro-enteritis was relatively high among artificially fed infants and relatively low among breast-fed infants.

(iv) That of the small number of breast-fed infants suffering from gastro-enteritis, the great majority was suffering from some severe parenteral infection.

The value of breast milk as the ideal natural food for the infant, its freedom from infection and the increased resistance to all disease processes possessed by the breast-fed infant are widely recognized. This strong protection afforded by breast milk feeding is possibly the result of many factors.

It is one of the purposes of this paper to suggest that the maintenance of the acid reaction and of the homogeneous, Gram-positive flora of the breast-fed infant's intestinal contents constitutes a most important underlying factor of this protection.

The following facts which have been established in this present study, would seem to support this suggestion.

The new born infant from one to twenty four hours passes stools which are alkaline in reaction. This suggests a suitable medium for the growth of a mixed flora, but the infant has not been exposed to infection long enough and the stools are found to contain few or no organisms.

The infant of from two to three days passes alkaline stools containing numerous Gram-positive and Gram-negative bacillary and coccal forms. Various organisms from the infant's surroundings have entered through his nose, mouth and anus and have found a suitable medium for their growth and proliferation.

After ten to twenty-one days of breast feeding the stools become acid in reaction and the typical homogeneous Gram-positive flora appears. The infant at this age is still receiving various organisms through his nose, mouth and anus, but the medium is not suitable; the nursling flora predominates, remaining constant and unaffected by the invaders. Not only are these invaders resisted, but the previous invaders who thrived in the alkaline fæces of the two to three day old infant, have been ousted.

A breast-fed infant suffers from some severe parenteral infection, his stools develope an alkaline reaction and their typical nursling flora is replaced by a predominantly Gram-negative mixed infection. It seems logical to suggest that it is possibly the loss of the protection against extraneous infection afforded by an acid reaction and a nursling flora of the fæces, as well as a general lowering of resistance to disease (which is the usual teaching) which leaves these infants prone to intestinal disturbances.

With the introduction of the more common artificial foods, the characteristic nursling flora and hydrogen ion concentration are lost, the stools become alkaline in reaction and a mixed, predominantly Gram-negative flora appears. These infants are now prone to gastro-enteritis and infections with organisms of the *Bacillus dysenteriæ* type.

The great majority of the stools of infants suffering from gastro-enteritis is alkaline in reaction and the flora a mixed one, Gram-negative types predominating.

In both of the last two conclusions the apparent protection of the acid reaction and nursling flora

of the fæces has been lost and many extraneous organisms are thriving in an alkaline medium.

As regards infection with *Bacillus dysenteriæ* it seems that this typical nursling hydrogen ion concentration and flora may again afford protection. This supposition is supported by the following statements.

Kendall contends that the dysentery bacillus seldom passes beyond the intestines and Todd has shown that it cannot elaborate its toxins in an acid medium.

Kendall has also shown that one of the essential accompaniments of an infection with *Bacillus dysenteriæ* is a putrefactive flora (which means an alkaline reaction of the fæces).

The Medical Research Committee in the experiments upon the isolation of dysentery bacilli in fæcal material of different reactions, found that these bacilli could not be detected in fæces having an acid reaction (approximately equal to that of nursling fæces) after even a few hours' exposure.

Possibly these facts and their underlying principles, although probably of little or no avail in the treatment of the established disease, could be utilized as valuable prophylactic measures against gastro-enteritis.

It would seem logical to suggest that if by some means the acid reaction and characteristic flora of the nursling stool could be maintained until the child was nearly nine months of age, a considerable diminution in the incidence of gastro-enteritis would be achieved. Possibly a diminution in the mortality rate amongst those relatively few infants who develope gastro-enteritis, would also be produced by these means.

In the totally breast-fed infant Nature has provided for the maintenance of this acid reaction and characteristic flora.

Can some artificial food formula be invented with which, when necessary, breast milk feedings could be complemented or replaced without destroying the apparent protection afforded by the reaction and flora of the nursling stool?

This question, at present unanswered, is possibly of great importance to Australia and other countries as a possible means of combating gastro-enteritis, diminishing its incidence and its mortality rate.

It is not the purpose of this paper to discuss the various theoretical possibilities in this connexion. But, having suggested the value of such a formula, two very brief experiments have been carried out in the hope of throwing some light on the subject.

Three infants (between three and five months of age) receiving a diet of modified dried milk, were given two cubic centimetres of a living culture of *Bacillus lactici acidi* three times daily. One infant received these feedings for nearly five weeks and the other two infants for approximately three weeks. There was no apparent increase in the acidity of the stools and no obvious change in their flora was noted (see Table IX).

Table IX.

A tabulated report of the stools from three infants to whom two cubic centimetres of a culture of *Bacillus lactici acidi* were fed three times daily. These infants were under Dr. Margaret Harper's care in the Medical Wards of the Royal Alexandra Hospital for Children, and were suffering from malnutrition.

Case I.—E. M., four months old; diet: modified cow's milk.

Day.	pH Value of Stools.	Naked Eye Characteristics of Stools.	Gram stained Stool Smear: Predominance.
1	7·0	Pale yellow, paste-like	Gram-negative.
2	Feedings of *Bacillus lactici acidi* commenced		
4	7·25		
7	7·5	Pale yellow, semi-fluid	
10	7·5	Pale yellow, semi-fluid	Gram-negative
14	6·5	White, paste-like	
21	8·0		
34	7·5	Greenish-white, paste-like	Gram-negative.

Case II.—F., twelve weeks; diet: dried milk formula.

Day.	pH Value of Stools.	Naked Eye Characteristics of Stools.	Gram stained Stool Smear: Predominance.
1	7·5	Yellow and semi-formed	Gram-negative.
2	Feedings of *Bacillus lactici acidi* commenced		
2	8·0	Yellow and formed	
9	7·0	Yellow, paste-like	Gram-negative.
16	6·75	Pale yellow, paste-like	
23	8·0	Pale yellow, fluid	Gram-negative.

Case III.—M., five months; diet: modified cow's milk.

Day.	pH Value of Stools.	Naked Eye Characteristics of Stools.	Gram stained Stool Smear: Predominance.
1	8·0	Yellowish-white, fluid	Gram-positive? Gram-negative?
3	Feedings of *Bacillus lactici acidi* commenced		
7	6·5	Yellow, fluid	
10	7·5	Yellow, semi-formed	Gram-negative.
17	7·5	Yellow, paste-like	
25	7·0	Yellow, semi-fluid	Gram-negative.

Feedings of a Lactic Acid Milk Formula.

Following the teaching of several American observers, it was supposed that feedings of a lactic acid milk formula would result in acid stools and possibly a flora similar to that of the nursling.

The lactic acid milk formula is two medicinal tablespoonfuls of "Lactone Syrup," containing 33·63% of dextri-maltose, 25·6% of dextrin and 6·5% of lactic acid (British Pharmacopœia) in 568 cubic centimetres (one pint) of cow's milk. This contains approximately 7·9% of carbohydrate, 3·5% of fat and 3·5% of protein.

But it was found that even after seven weeks of lactic acid milk feedings, infants continued to pass stools with an alkaline reaction and a variable flora in which Gram-negative types predominated.

Of the eleven specimens examined the average pH value was found to be 7·45 and a predominance of Gram-negative flora was noted in approximately 72% (see Table X).

Breast Milk Feedings Complemented with Feedings of a Lactic Acid Milk Formula.

An examination of the stools of eight infants, receiving breast milk supplemented by lactic acid

milk, showed an average pH value of 6·9, a stool similar in appearance to that of a nursling in 75%, and a considerable predominance of gram-positive bacillary forms in 62·5% (see Table XI).

TABLE X.

Infants Fed on a Lactic Acid Milk Formula.

Age, Months.	Approximate Duration of Lactic Acid Milk Feeding, Weeks.	Naked Eye Appearance of Stools.	pH Values.	Flora.
4	4	Nursling type	7·5	Mixed flora, slight predominance of Gram-negative forms.
3	5	Pale yellow, semi-liquid	8·0	Gram-negative bacilli predominant.
6	3	Nursling type	7·5	Mixed flora, Gram-negative predominance.
3½	2	Nursling type	7·0	Gram-positive bacilli (large and small) predominant, Gram-negative bacilli and cocci.
5	7	Dry and chalk-like	8·0	Mixed flora, Gram-negative predominance.
3	2	Nursling type	6·5	Gram-negative predominance, numerous Gram-negative bacilli and cocci.
2	2½	Yellow, semi-liquid	8·0	Gram-positive bacilli (large) and Gram-negative bacilli (large and small) in about equal proportions.
3	4	Nursling type	7·5	Predominance of small Gram-negative bacilli, Gram-positive and Gram-negative bacilli and cocci present.
5	2	Nursling type	8·5	Predominance of Gram-negative bacilli (large and small).
4	3	Nursling type	5·5	Gram-positive bacilli (large and small), Gram-negative bacilli and cocci.
5½	5	Firm, dry and pale	8·0	Mixed flora, slight predominance of Gram-negative forms.

TABLE XI.

Breast Milk Feedings complemented with Feedings of a Lactic Acid Milk.

Age, Months.	Approximate Duration of Complementary Feedings of Lactic Acid Milk, Weeks.	Naked Eye Appearance of Stools.	pH Value.	Flora.
3	6	Similar to nursling stool	7·0	Numerous large and small Gram-positive bacilli, a few small Gram-negative bacilli and cocci.
5	4	Similar to nursling stool	7·5	Large Gram-negative bacilli predominant.
1½	3	Pale stool, similar to nursling stool in consistency	5·5	Field almost obscured by large Gram-positive bacilli.
3	2½	Nursling type	6·5	Predominance of Gram-positive bacilli, some Gram-negative coli-form bacilli and Gram-negative cocci.
6	7	Dry, pale yellow stool	7·5	Gram-positive and Gram-negative bacilli in about equal numbers, Gram-positive and Gram-negative cocci.
3	3	Nursling type	6·5	Large Gram-positive bacilli predominant, Gram-negative bacilli and cocci present.
4	2	Dry and hard pale stool	8·0	Gram-negative predominance, mixed flora.
5	3½	Nursling type	6·5	Predominance of large and small Gram-positive bacilli.
2	2	Pale, semi-formed stool	7·0	Gram-negative predominance, chiefly large bacilli.

In contrast to these results, a study of the stools of six breast-fed infants receiving complementary feedings of a cow's milk or a dried milk formula showed an average pH value of 7·6 and a Gram-negative predominance in 60% (see Table XII).

TABLE XII.

Breast Milk Feedings complemented by Feedings of Cow's Milk or Dried Milk Formula.

Age, Months.	Approximate Duration of Complementary Feedings, Weeks.	Diet.	pH Value of Stools.	Flora.
3	3½	Breast milk and modified "Lactogen"	7·5	Mixed flora, Gram-negative predominance, many Gram-positive bacilli present.
4½	4	Breast milk and modified cow's milk	8·0	Gram-negative predominance.
3½	4	Breast milk and modified "Lactogen"	7·5	Predominance of Gram-positive bacilli, a few small Gram-negative bacilli.
5	8	Breast milk and modified "Lactogen"	8·0	Gram-negative predominance.
2½	3	Breast milk and "Prescription Glaxo"	7·0	Gram-positive bacilli (large and small), some Gram-negative bacilli and cocci.
6	9	Breast milk and modified cow's milk	8·5	Gram-negative predominance, mixed flora.

The question of establishing a food formula which would afford protection by preserving the characteristic nursling flora and acid reaction, remains unanswered.

Both the feeding of living cultures of *Bacillus lactici acidi* and the lactic acid milk feedings proved to be unsatisfactory in this connexion. But the results obtained by complementing breast milk, when deficient, with lactic acid milk feedings suggest that this procedure might be of value as a means of preserving what is assumed to be the nursling's natural protection against gastro-enteritis.

Acknowledgements.

I should like to express my thanks to the members of the honorary medical staff and Medical Superintendent of the Royal Alexandra Hospital for Children for permission to make use of patients in the hospital, to Dr. Margaret Harper for permission to make use of infants at "Tresillian," to Dr. F. Tidswell for his help and suggestions, to Dr. Bruce Williams for his help in obtaining specimens from new born infants and to the sisters and nurses who have helped me to carry out this study.

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DIFFERENTIAL DIAGNOSIS OF WASTING IN THE INSANE.

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Anyone who has had experience of work in a mental hospital will have had to face the problem of the rapid wasting which occurs in young adult patients. What is the cause of the wasting? Is it due to tuberculosis, to diabetes, perhaps to sulphonal poisoning or is it simply due to the mental disorder? The type of case which gives rise to this difficulty of diagnosis, is that of the patient between twenty and thirty years of age, who on admission is in good general health, weighing, say, sixty-four kilograms or ten stone, but who in a year's time weighs no more than thirty-two kilograms and shortly afterwards dies in an emaciated condition. The patients are usually of the *dementia præcox* type, often excited, catatonic, resistive especially to feeding and degraded. They have usually required treatment with such drugs as sulphonal, chloral or paraldehyde and are almost invariably extremely difficult to examine by ordinary bedside methods.

When once wasting of this type has been noticed and duly reported, the medical officer wants to know whether he must place the patient's name on the list of tuberculous patients; whether the patient should be moved to a ward specially devoted to the treatment of the tuberculous; whether the patient is receiving an adequate diet; whether artificial feeding is necessary; whether the patient has been too heavily drugged or whether the patient is suffering from such a wasting disease as diabetes. Sometimes the wasting is due to chronic nephritis and these patients develope a terminal diarrhœa which may be mistaken for dysentery and so cause trouble for administrator and clinician alike.

These problems and others have to be faced in cases of this sort and the cases are by no means uncommon. It is galling to have to report to the authorities that patient X died of pulmonary tuberculosis of a year's duration, although he was not regarded as tuberculous before autopsy or on the other hand to report that patient Y died of *dementia præcox*, although prior to *post mortem* examination the condition was regarded as actively tuberculous. Anyhow, we do not like making a mistake, even though we alone know of the error. The object of this article is an attempt to systematize the differential diagnosis of the wasting, to point out some of the difficulties and to show how some simple laboratory tests can help in reaching a diagnosis which will stand the test of autopsy. The opinion is expressed that if patients with wasting are examined on the lines of this paper, the diagnosis will not err.

The causes of wasting are: (i) starvation and malnutrition, (ii) pulmonary tuberculosis, (iii)

diabetes, (iv) hyperthyreoidism, (v) chronic nephritis, (vi) chronic poisoning, (vii) *dementia præcox*.

There is no need to include in this list wasting due to malignant growths because the diagnosis of that condition is the same in the insane as in the sane.

It will probably be noticed that general paralysis of the insane has not been included in the above list, although rapid wasting is one of the most striking features of the so-called fulminating form. The cause of the omission is that general paralysis of the insane would be detected probably on the first clinical examination and at any rate after the arrival of the report on the Wassermann test, which (as is now generally the practice) should be performed on all new patients.

Wasting Due to Starvation or Chronic Malnutrition.

Lack of food resulting from preoccupation with hallucinations, stupor, persecutory delusions or suicidal tendencies may cause wasting. When the patient takes to a complete fast the condition is easily detected and treated. Patients are able, however, by evading observation, to take a subnormal diet. These patients waste in consequence. The detection of acetone in the urine of such patients is a great help in diagnosis. Moreover, as the acetone can easily be estimated by a rough quantitative method, the amount will serve as an indication of the degree of starvation and also, if estimations are repeated, of the progress of the case with treatment. It has been found that the presence of acetone in the urine is a useful indication for artificial feeding in cases of chronic underfeeding amongst resistive melancholics and others.

Wasting Due to Pulmonary Tuberculosis.

At first it would appear that anyone of average clinical skill could diagnose by means of symptoms and physical signs such a condition as severe pulmonary tuberculosis. This, however, is not the case with the insane; the following are some of the reasons for the difficulty of diagnosis.

(i) The cough which among the sane is a very important symptom, is very inconstant among the insane and at times misleading. A patient with *dementia præcox* may have advanced active pulmonary tuberculosis with no appreciable cough. On the other hand, a patient may have a severe cough with or without sputum, not dependent upon any tuberculous lesion in the lungs. These coughs are sometimes in the nature of mannerisms and sometimes due to irritation resulting from rhinitis and pharyngitis, common conditions in the stuporous.

(ii) The temperature chart may be a valuable indication. A patient with *dementia præcox* may start his downhill course with an attack of tuberculous broncho-pneumonia, having continuous severe pyrexia and signs of patchy consolidation. These patients may survive the acute stage and then pass on to an active chronic condition. In others with *dementia præcox*, however, extensive tuberculous lesions in the lungs may be found *post mortem* in spite of the fact that for months before death the patients have remained apyrexial. There are other non-tuberculous patients whose temperature charts, though rather difficult to interpret, are not unlike the charts of tuberculous patients. The pyrexia in these circumstances is probably due to toxic absorption associated with constipation.

(iii) Sweating may be a valuable guiding symptom, though its value is somewhat impaired by the frequency with which the mentally disordered sweat from other causes than pulmonary tuberculosis. Sweating may be caused by agitation, fright, hallucinations and masturbation, all likely to occur at night, especially in *dementia præcox* of the wasting type. The issue is thus confused.

(iv) Careful physical examination of the patients like those under consideration (wasting, *dementia præcox*) is often an extremely difficult, if not an impossible task. The patient sits huddled up, with the pectorals contracted and possibly twitching, the chin pressed tightly against the chest, so tightly sometimes as to cause a bruise; there is continuous dribbling and probably sucking noises are being made with the tongue and the cheek or the teeth are being ground together. All the time the patient's breathing is so shallow that it is hard to imagine how such a small respiratory exchange can supply sufficient oxygen to the tissues. In these circumstances the results of physical examination are practically valueless and it is only in exceptional conditions that the stethoscope is of any service.

(v) Not infrequently patients are seen in whom the wasting is associated with brown pigmentation of the skin, low blood pressures, progressive weakness and possibly some pyrexia and cough. These conditions, though often diagnosed as Addison's disease, are generally found not to be associated with tuberculous lesions of the lung or suprarenal at the *post mortem* examination. The cough is often due to chronic bronchitis which like the pigmentation may be the result of arsenical administration. The wasting and other signs seem to be associated with the mental disease.

(vi) It is on the laboratory tests that most reliance can be placed. If the patient cannot or will not cough into a vessel provided, sputum may sometimes be obtained by the artificial induction of coughing by local irritation and then by collecting the coughed up material on a swab of suitable size. The author (probably due to lack of skill) has not been very successful in his "fishing for sputum" efforts. If the sputum is submitted to some concentrating method (for example, by the "Antiformin" method) before staining, both positive and negative findings are of value. If only a direct smear is taken, failure to demonstrate tubercle bacilli is of little value.

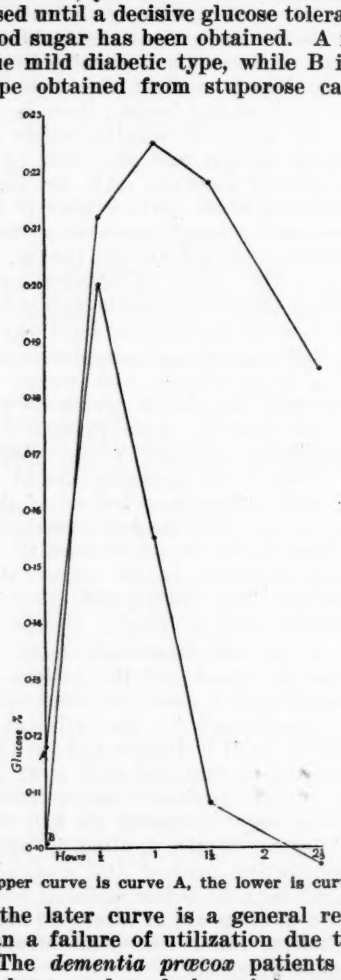
When sputum is not obtainable, the fæces should be examined by some concentrating method. Tubercle bacilli pass through the alimentary canal without losing their staining properties and apparently the presence of acid and alcohol fast bacilli in the fæces indicates tuberculosis without doubt.

The author has found in a series of nearly one hundred patients known to be suffering from tuberculosis and controls that the results of fæces examinations are rather more reliable than those of sputum examination. Similar results have been obtained in institutions for children.

Mental hospitals do not as a rule have X ray installations available for radiography of the chest. Therefore, this aid to diagnosis has not been considered.

Wasting Due to Diabetes Mellitus.

It may be found either as a result of a routine examination or in the course of the investigation of the wasting that the patient has glucosuria. Often tests repeated on subsequent occasions do not show the presence of sugar. Sugar may be present on several occasions, yet *diabetes mellitus* should not be diagnosed until a decisive glucose tolerance curve of the blood sugar has been obtained. A is a curve of the true mild diabetic type, while B is a curve of the type obtained from stuporose cases. The



The upper curve is curve A, the lower is curve B.

defect in the later curve is a general retardation rather than a failure of utilization due to lack of insulin. The *dementia præcox* patients are also liable to have a low leak point, resulting in glucosuria with a blood sugar value of less than 0·17%. Some of these patients are real diabetics and often of the fulminating type. It is reported that some derive considerable benefit from treatment. Rare cases of hæmochromatosis (bronze diabetes) are met with; these patients, in addition to diabetes, have bronze pigmentation and liver and kidney defects.

Wasting Due to Hyperthyreoidism.

Closely allied to the above conditions from the point of differential diagnosis is severe hyperthyreoidism, Graves's disease, with little or no exophthalmos. The patients have wasting, sweating, tachycardia, occasional pyrexia, occasional glycosuria, some pigmentation, possibly slight enlargement of the thyreoid and a degree of

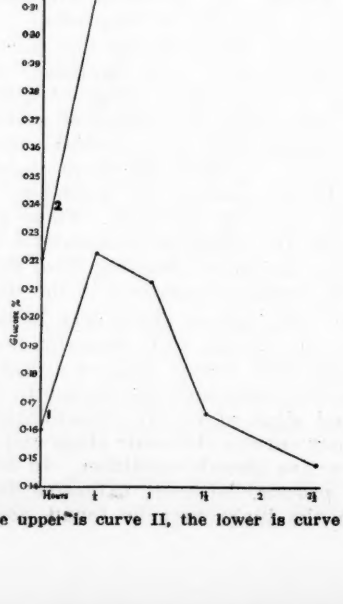
Table I.

Time in relation to the Ingestion of Fifty Grammes of Glucose.	Blood Sugar Content.	
	Curve A.	Curve B.
	%	%
Before	0·118	0·101
½ hour after	0·201	0·213
1 hour after	0·225	0·155
1½ hours after	0·219	0·108
2 hours after	—	—
2½ hours after	0·185	0·097

Patient A was a mild diabetic.
Patient B was a stuporose patient.

exophthalmos. In the absence of exophthalmos and enlargement of the thyreoid this condition is difficult to diagnose from Addison's disease and (owing to the glycosuria) from diabetes. They present a typical glucose tolerance curve, have tachycardia and their mental condition is different from the usual type of wasting patient; they have more insight, are unduly apprehensive, querrulous and sometimes acutely depressed and suicidal.

It is of interest that these patients occasionally develope diabetes, possibly as a result of the prolonged strain on the pancreas. The author has



The upper is curve II, the lower is curve I.

seen one. Curves I and II indicate how within the space of three weeks the condition of diabetes had set in.

Table II.

Time in relation to the Ingestion of Fifty or Twenty Grammes of Glucose.	Blood Sugar Content.	
	On November 27. Curve I.	On December 18. Curve II.
	%	%
Before	0·160	0·221
½ hour after*	0·223	0·320
1 hour after	0·213	0·336
1½ hours after	0·166	0·338
2 hours after	—	—
2½ hours after	0·148	0·318

* On November 27 fifty grammes of glucose were given and on December 18 only a few days before patient's death twenty grammes.

Wasting Due to Chronic Nephritis.

A certain number of wasting patients have some albuminuria and some die of uræmia. The author has heard of one whose blood was examined in the course of a research on creatinine in the blood. The percentage of creatinine was found to be particularly high. A few weeks later the patient (a young female) died of uræmia. If albumin is detected in the urine of a wasting patient, the centrifugalized deposit should be examined for casts. To insure that the albumin is not of the non-pathogenic type (for example, static) a blood urea estimation should be made. Either the method of Ambard, which requires about ten cubic centimetres of blood and is only approximate in the results, or that of Twort (modified) which requires only 0·2 cubic centimetre of blood and is reasonably accurate, can be used. The best possible test for true kidney defect (for reasons to be given hereafter) is a blood urea curve with the latter technique used for each estimation.

Briefly, the test is carried out as follows. Blood is taken in the early morning in order to secure the "fasting" blood urea. A meal of urea (usually fifteen grammes) is then given and subsequently specimens of blood are taken at half-hourly intervals for three hours. When the urea contents of the different fractions have been estimated, the results are plotted and a curve obtained. The curve is, in the opinion of the author, more reliable than the usual urea concentration test.

Wasting Due to Chronic Poisoning.

Some patients, owing to their mental condition, excitement and violence, require heavy doses of bromides, chloral and sulphonal, perhaps 3·6 grammes (sixty grains) of bromide, 1·8 grammes (thirty grains) of chloral and fourteen cubic centimetres (four fluid drachms) of paraldehyde during the twenty-four hours. When these patients waste, the drugs must be considered as at any rate a factor in the process. Pulmonary tuberculosis occurs not infrequently in these necessarily heavily drugged patients, but its diagnosis from wasting due to drugs alone is difficult and depends almost entirely on laboratory findings. These patients are liable to develope pulmonary œdema, broncho-pneumonia and pyæmia, conditions which obscure the true diagnosis.

Really chronic sulphonal poisoning is an interesting condition which may be discussed at this point. Acute sulphonal poisoning is easy to detect with its collapse, diarrhœa and hæmatoporphyrinuria, but chronic poisoning, due to taking perhaps 1·2 grammes (twenty grains) twice a day for many months, is a condition which is less frequently described. The patients may have some or all of the following symptoms:

The skin generally and particularly the skin of the hands and feet becomes ichthiotic, hard and branny.

The hair becomes brittle and tends to fall out. The hair also acquires an unnatural pigmentation; it looks as though it had been dyed.

On exposed parts, such as the hands, forearms and face, there may be in sunny weather a severe dermatitis. A dermatitis has been produced experimentally in animals by exposure to the sun or to ultra-violet rays after subcutaneous injection of hæmatoporphyrin.

The eruption may resemble typical pellagra.

Comedo of the nose and "bat's wing" area of the face may appear. The long brittle plugs which fill the sweat glands, have a characteristic brownish-yellow appearance.

Stomatitis which may be severe, is not an uncommon development.

Noma vulvæ has been observed

Diarrhœa is a common late symptom and if other more obvious signs have not developed or been detected, this condition may be mistaken for tuberculous enteritis or dysentery.

The urine usually contains an excess of urobilin. The liver function tests give results showing some impairment of function.

Hæmatoporphyrinuria, though obvious in the acute poisoning, is more difficult to detect in chronic poisoning.

Sometimes even after the drug has been discontinued the patients may be seized with syncopal attacks in one of which they die. In one patient most of her symptoms appeared after sulphonal had been omitted. She had had two or three syncopal attacks from which she recovered, and she appeared to be progressing favourably until one morning while the author was in the ward preparing to commence a lævulose tolerance test, she had another syncopal attack and died.

To return from this digression, it will be well to remark that the differentiation of chronic poisoning from the other causes of wasting depends upon clinical observations and such laboratory tests as have been indicated.

Wasting Due to Dementia Præcox.

There are wasting patients whose symptoms and signs during their downhill course have suggested some or all of the previously considered causes of

wasting, yet at autopsy no lesions are found. The patients just fade away like the old soldiers of the popular ditty. As the end approaches the patients only "tick over," the pulse may be impalpable at the wrist for some days before death and the respiratory rate slows down to five or even one in the minute and finally one shallow breath every two minutes seems to suffice to keep them alive for an hour or so. *Post mortem* examination reveals no gross lesion in any of the organs. The endocrine glands are small and atrophic and the heart and great vessels are usually small and almost infantile, the heart showing vitreous degeneration. The early diagnosis of these conditions seems to depend entirely upon negative findings, yet if the other investigations have been thoroughly carried out, the correct diagnosis can be made early and, if death occurs, the autopsy can be faced with confidence. The cause of death is usually given as *dementia præcox* or (if such terms apply) exhaustion from melancholia, though the certificate is sometimes "strengthened" by the inclusion of vitreous degeneration of heart or broncho-pneumonia, which when present is of the hypostatic variety.

If more were known of the nature of the changes involved in this "fading away" phenomenon, it would probably be easier to discover the cause of *dementia præcox*.

Acknowledgement.

The author is indebted to Dr. J. Bentley, Inspector-General of the Insane, for permission to publish this paper and to Dr. E. J. T. Thompson and Dr. G. Bury for their advice and assistance.

Reports of Cases.

SUPERFŒTATION.

By Rupert A. Willis, M.D. (Melbourne),
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De Lee in his "Principles and Practice of Obstetrics," states:

> Superfetation is the nesting of a second fetus in the uterus already occupied by one in the process of development. Its occurrence has been held possible by American and French authors, while the English and German writers usually deny it. A double uterus may carry a child in each compartment, as in the cases quoted by Ramsbotham—one, where a woman, five months and sixteen days after the delivery of a living seven months' child, delivered another of full development, and another where two well-formed, fully developed boys were born three months apart. These cases should not be called superfetation. Since the *decidua reflexa* does not unite with the *vera* until the middle of the fourth month, and since ovulation occurs during pregnancy, theoretically superfetation is possible, but in most instances cannot be proved. Differences in the size of the fetuses may be explained by unequal development, but F. T. Andrews, Barry, and the author have seen specimens which prove the occurrence of superfetation in the human female. Dr. Zimmerman, of Cameron, Illinois, found a healthy ovum of four weeks, together with a healthy fetus of ten weeks, in an aborted mass.

As De Lee points out, those who deny the occurrence of superfœtation, explain away alleged examples as due either to double uterus or to unequal development of twins. But, as De Lee states, specimens, like Dr. Zimmerman's, showing two normal fœtuses of different ages in a single aborted mass definitely establish that true superfœtation occurs. The object of this article is to describe in detail just such a specimen, containing normal fœtuses of nine and sixteen weeks respectively.

A woman aged thirty-five, a *multipara*, with three children, twelve, eleven and ten years of age, had had amenorrhœa for four months. The first day of her last menstrual flow had been April 9. On August 6, while dancing, she "felt something give away," and passed a

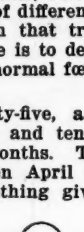

Figure I.
Sixteen Weeks Fœtus, Half Natural Size.

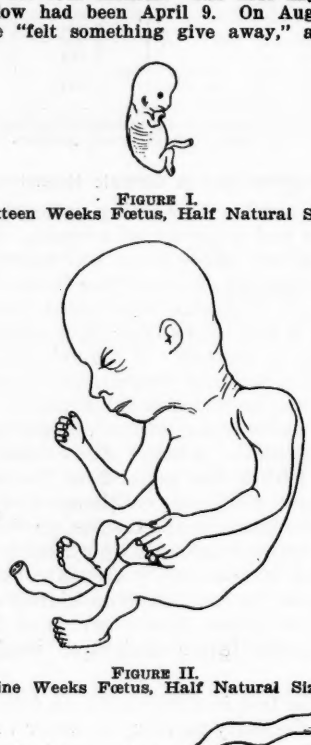

Figure II.
Nine Weeks Fœtus, Half Natural Size.

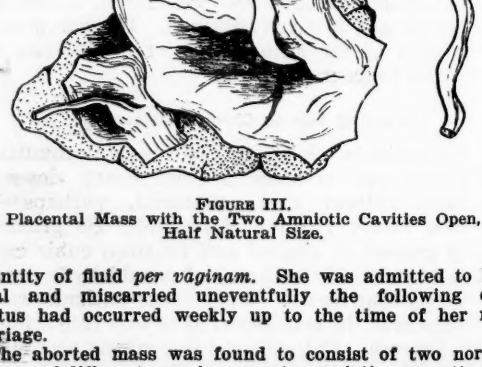

Figure III.
Placental Mass with the Two Amniotic Cavities Open, Half Natural Size.

quantity of fluid *per vaginam*. She was admitted to hospital and miscarried uneventfully the following day. Coitus had occurred weekly up to the time of her miscarriage.

The aborted mass was found to consist of two normal fœtuses of different ages in separate amniotic sacs, attached to a common mass of placental tissue (see figures I, II and III).

The larger fœtus measures 17·5 centimetres standing height, and 12 centimetres sitting height. Its age is there-

fore sixteen weeks.[1] The skin is transparent, with cutaneous vessels showing plainly through it; early lanugo hair is appearing; the eyelids are fused; the nails are well formed; the external sex organs, male, are well differentiated. These are the normal characters of a sixteen-weeks fœtus. The ossification of the skeleton as seen in a skiagram is that of a normal fœtus of sixteen weeks. The umbilical cord is 27 centimetres long and of an average diameter of seven millimetres; the amniotic sac is incomplete.

The smaller fœtus measures 31 millimetres long and is therefore nearly nine weeks old,[1] an age confirmed by the following features. The nose, mouth and external ear are well formed; there are no visible traces of the branchial arches; the eyelids are fused; the limb buds show three easily distinguished segments and the rudiments of digits; the caudal appendage has disappeared; there is no sex differentiation in the rudimentary external genitals; the umbilical orifice is contracted; the umbilical cord measures 20 millimetres in length and 1·5 millimetre in diameter and is conically expanded at its amniotic attachment. The amniotic sac is ruptured, but evidently fairly complete; it is quite distinct from the larger sac and in its unruptured state would have measured roughly 2·5 centimetres in diameter. A skiagram shows conspicuous ossification of the whole shaft of the clavicle and of the rami of the mandible; well marked ossific centres in the maxillary region of the face, in the shafts of the femur, tibia and fibula and to a less extent in the long bones of the upper limb; well advanced ossification of the ribs is present. These features are those of a normally developed nine weeks fœtus.

The specimen described thus consists of a single mass of placental tissue, attached to which are two separate amniotic sacs, containing fœtuses. By external features and skeleton ossification these are clearly perfectly healthy specimens of sixteen and nine weeks' development respectively. Double uterus is obviously out of the question and it is highly improbable that "unequal development" of dichorionic or double-ovum twins would result in such typically normal fœtuses.

This specimen therefore, like those mentioned by De Lee, definitely proves the occurrence in the human being of the exceedingly rare phenomenon of true superfœtation.

Acknowledgement.

I am indebted to Dr. Hall of Eltham for permission to describe this specimen, which he kindly presented to me.

Reference.

[1] Arthur Keith: "Human Embryology and Morphology," Fourth Edition, 1921, page 51.

LARGE CONGENITAL UMBILICAL HERNIA.

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The very large size of the hernia, the apparent hopelessness of the condition and yet the complete recovery following operation, justify the following report.

Clinical History.

On June 27, 1928, Mrs. F. after a normal confinement, gave birth to a male child weighing about 3·15 kilograms (seven pounds). The portion of the umbilical cord adjacent to the abdominal wall was enlarged to almost the size of a fœtal head. The size of the opening in the abdominal wall at its junction with the hernia, was about 11·25 by 6·25 centimetres (four and a half by two and a half inches). Through the transparent sac wall could be seen the whole liver and the larger and small bowel. The abdomen appeared to be quite empty and the abdominal wall on either side of the hernia was quite flattened, as apparently the greater part of the intestinal tract and liver had remained outside the abdominal cavity. The cord was attached to the apex of the somewhat conical shaped hernia. The condition appeared to be a hopeless one, but as there were no other abnormalities present, it was decided to give the infant the very slender chance that an operation seemed to offer. The operation was done twelve hours after birth, ether being administered by Dr. F. H. Gaden, who referred the patient to me.

Under the anæsthetic an attempt was first made, by pinching the hernia from above downwards with fingers, bowel clamps and sponge forceps, to see if it were possible to force the contents of the hernia into the abdomen. Under this pressure continued for five or six minutes the hernia was forced into the abdomen, but could be retained there only by keeping up constant pressure and when the hernia was inside, the abdominal wall was as tense as a drum. The coverings of the hernia consisted of amnion and peritoneum which appeared to be fused together, and some Wharton's jelly. It was surprising the amount of pressure the sac stood without rupturing. As I felt that if I opened the sac before placing some of the sutures, I might not be able to keep the bowel back, an incision was made first through the margin of the skin, close to the amniotic covering of the sac and care was taken not to open the peritoneum, and this incision was continued right round the hernia. Silkworm gut sutures were then inserted through the skin about 1·25 centimetres (half an inch) from the cut edge. An endeavour was made to pick up as much tissue as possible without opening the sac. These sutures were carried over the hernia and inserted in the reverse direction on the other side. After having placed about five or six sutures close together over the lower end of the hernia, I treated the upper end in a similar manner. The sac was then opened and cut away as the sutures were tied. A few sutures at the middle of the wound were made to penetrate the whole thickness of skin and peritoneum. It was very difficult to tie the sutures without nipping the bowel and at the lower end of the incision there was difficulty in keeping the bladder back whilst the sutures were being tied. The operation took about three-quarters of an hour and it was decided to leave the skin coaptation sutures for a couple of days, if the child survived. The wound was well supported by strapping placed right round the child.

The infant stood the operation well and seemed quite normal next day. On the third day as the child was still progressing I decided to unite the skin edges with horsehair and at the same time I placed about six more silkworm gut sutures outside the others. This was done without an anæsthetic. On inserting these silkworm gut sutures care was taken not to penetrate the peritoneal cavity, the needle taking up little more than skin on either side. These sutures were secured by clamping them on either side of the wound with split lead shot, a flat piece of rubber being interposed between the skin and lead. This procedure was resorted to as it was feared that the first layer of sutures might give way owing to the distension of the abdomen. On the fifth day there was an escape of a very slight amount of fæcal matter from the wound, but curiously enough there was no recurrence of this. All the sutures were removed by the sixteenth day when the wound was soundly healed. The child took the breast from the first day and every week has shown a good gain in weight. It is now over three months since the operation and it appears normal in every way with the exception of the bulging of the skin along the incision, where, of course, there is only skin union. A belt made of "Z.O." strapping is kept on constantly to prevent the skin from yielding any more. It should be possible, when the child is a year or more old, to bring the muscle and fascia together and restore the abdominal wall to its normal condition.

Comment.

Although this condition is generally called hernia, it is not a true hernia because the contents of the sac have never really been inside the abdomen, having developed in the cord. Hence the difficulty in cases like the one recorded in putting the contents of the sac into the abdominal cavity. In the above case the ligaments of the liver were attached to the interior of the sac and had to

be divided when the sac was removed. I have searched the available literature for records of similar cases, but can find no reference to a patient with such a large exomphalos recovering after operation. I recorded a case myself in THE MEDICAL JOURNAL OF AUSTRALIA[1] in which the whole liver was in the cord and the infant made a good recovery after operation, but in this case the actual size of the opening in the abdominal wall, was about 3·75 centimetres (one and a half inches) in diameter and it had to be enlarged before the liver could be returned, so that there was no difficulty in bringing the abdominal wall together. Ballantyne states that hernia of the umbilical cord occurs about once in every five thousand births.[2]

References.

[1] P. L. Hipsley: "Liver in Umbilical Cord of New-Born Infant," THE MEDICAL JOURNAL OF AUSTRALIA, October 17, 1925, page 484.

[2] Ballantyne: "Antenatal Pathology and Hygiene," 1904, page 524.

Reviews.

A BOOK ON DIAGNOSIS.

THE writing of a book on surgical diagnosis is far from easy, for the limitation of the term must be exceeded owing to the necessity for some description of the various diseases, injuries and deformities. The realm of the physician, too, must be at times encroached upon. Ætiology, pathology, symptoms and signs, the personal, family and clinical history and even the effects of treatment may be called upon to play their respective parts in the elucidation of the various problems that face the diagnostician. In his text book for busy practitioners and surgeons A. J. Walton has preserved a fair balance in the effort to bring the standard of surgical diagnosis up to modern requirements.[1]

With the exception of Kanavel, whose classical work on "Infections of the Hand" is world renowned, the editor, Mr. A. J. Walton, and his collaborators are representatives of the British schools of medicine. In the compilation of the work the editor has been ably assisted by what may be termed the younger school of British surgeons and the wisdom of his selection is evident in the number of well known contributors. Criticism of a wonderfully compressed work of this kind, teeming with an accumulation of reliable data from scientific sources and collated by surgeons eminent in their profession, resolves itself rather into admiration and gratitude for the information provided. With the exception of diseases of the eye, the field of surgical diagnosis is covered. Short sections, however, on subjects such as the ear, nose and throat are supplied by Patterson, of the London Hospital. The opening article on inflammation by Sir G. Lenthal Cheatle lays before the reader the difficulties in the connotation of the term and the liability to confusion with hyperplasia. In his later chapter, "Diseases of the Breast," he avails himself of the opportunity of dilating upon his practical work on the ætiology of breast tumours which reminds us of the wisdom of Ewing in entitling his well known book "Neoplastic Diseases." The use of laboratory methods is ably dealt with in a general way by Panton. Following this are helpful chapters on specific infections of various kinds, including tropical and venereal diseases.

Injuries, diseases and deformities of the different regions and organs are then dealt with in a manner becoming a work of over one thousand pages. Restriction of this space naturally precludes lengthy dissertation and discussion, but the practical application of methods of investigation is everywhere evident. The value of X rays in diagnosis is emphasized by the space devoted both in writing and illustration by numerous contributors to various subjects. The most modern diagnostic methods are greatly based upon the use of X rays in conjunction with opaque substances. In the diagnosis of gas gangrene the earliest sign, namely, the X ray evidence of gas bubbles in the tissues before they may be felt by the fingers, is not mentioned by the writer on that subject. The intentional production of crepitus in the effort to discover a fracture does not usually meet with the approval of the patient. If an X ray plant is available, why try to elicit this distressing sign? In the early diagnosis of perforated gastric and duodenal ulcers, when liver dulness is still present, there is no mention of the subdiaphragmatic collection of gas of crescentic form as described by Du Pasquier. The great value of the oral administration of the dye in cholecystography is accepted by Walton to the elimination of the dangerous intravenous method. In a newer edition he will probably prefer the not unpleasant solution of tetraiodophthalophenon by mouth to the bulky coated capsules or pills of sodium tetraiodophenolphthalein. The tracheal route for "Lipiodol" injection of the bronchus is much preferred to the more irritating crico-thyreoid one. Warning is given regarding the promiscuous use of injections of air in ventriculography with its 8% mortality, and of "Lipiodol" into the *cisterna magna*. These operations should be left in competent hands. In Victor Bonneys' article on matters gynæcological, the reader will miss reference to Rubin's work and to "Lipiodol" injection to determine the patency of the Fallopian tubes. These methods were freely discussed by him during his recent visit to Australia.

The book contains many notes of warning to young and enterprising surgeons. The now routine lumbar puncture should be performed with extreme care when the intrathecal pressure is high, because of possible herniation of cerebellum and medulla into the *foramen magnum*. Walton's article on visceroptosis should surely stay the hand of the habitual remover of vermiform appendices for pains in the right iliac fossa. The use of adrenalin in over strong solution might have been mentioned as a cause of gangrene, for example, in local anæsthesia. Lockhart Mummery, in a chapter written in his usual decisive way, draws attention to the early diagnosis of cancer of the rectum and strongly urges proper examination of the rectum in all cases of irregularity of the bowels and diarrhœa, especially in elderly people. In an excellent short article on diseases of the lungs and pleura Dr. Tudor Edwards advocates the use of the thoracope of Jacobaeus (Stockholm) as well as of X rays after the production of an artificial pneumothorax in the diagnosis of tumours of the chest. The instrument is introduced through a cannula into the pleural cavity after injection of a gas. Oxygen is preferred because of its rapid absorption and the absence of gas embolism.

For the benefit of ordinary surgeons we would suggest to Dr. Riddoch and Dr. Brain the inclusion of a few diagrams on cerebral localization to illustrate their admirable chapters on diseases of the brain and skull.

In spite of the number of contributors, overlapping is noticeable only in the sphere of injuries and diseases of the bones and joints dealt with by three separate writers. In attempting the separation of chronic *arthritis deformans* into two clinical types—rheumatoid arthritis and osteo-arthritis—Fisher wishes the distinction to be preserved "although borderland cases occur."

Sequiera in "Diseases of the Skin" confines the use of the word epithelioma to non-malignant tumours, the commonly accepted epithelioma being referred to as squamous carcinoma. Hydatid disease has a share in the book proportionate to English experience. The Casoni and complement fixation tests are mentioned, the former being attributed to Deusch.

Space forbids a detailed criticism of each chapter. In general the work reflects great credit upon the editor who has given to the profession a practically British exposition of the views of representative modern surgeons on diagnosis. The work is in two handy-sized volumes. The illustrations are numerous and well produced. Printer's errors are in places rather frequent: "extraversation" occurs more than once; "familiar" in hæmophilic regard becomes "familial" later on; "interperitoneal" obtrudes itself. "Lightening" (*sic*) pains in the rectum would no doubt be gladly borne by many sufferers. The capsule proper of the prostate gland is described as being of fibromyomatous tissue.

To those surgeons and practitioners who wish to consult a reliable work on surgical diagnosis within a reasonably compact space, we can heartily recommend these two volumes.

[1] "A Text-book of Surgical Diagnosis," Edited by A. J. Walton, M.S., F.R.C.S., B.Sc., M.B.; Volumes I and II; 1928 London: Edward Arnold and Company. Royal 8vo., pp. 1121, with illustrations. Price: 63s. net.

The Medical Journal of Australia

SATURDAY, APRIL 20, 1929.

The Chair of Obstetrics in Melbourne.

Early in December of last year the Council of the University of Melbourne issued a memorandum on the conditions of appointment to the chair of obstetrics and information with regard to the work. The history of the movement has been confused by rumours, contradictions and the exhibition of annoyance. According to Sir Edward Mitchell's account the Trustees of the Edward Wilson (*The Argus*) Trust held some time ago that certain concatenation of circumstances, aspirations and plans was so exceptional that they were justified in offering to the then Premier of Victoria the sum of £60,000 for the purpose of founding a chair of obstetrics to be called the Edward Wilson Chair, provided that the scheme of combining large hospital extensions with increased facilities for university medical teaching could be carried out in its entirety. We have no doubt that the late Edward Wilson would have endorsed this decision and would have derived satisfaction from the knowledge that some of his wealth would be employed for the purpose of improving the training of medical students in the science and practice of obstetrics and in the advancement of our knowledge of this subject. No one can deny that the safeguarding of the lives of expectant and parturient women and of their infants is a matter of transcendent importance to the nation. This is not a political matter and should not be influenced by the fortunes of any political party. If the gift would have been justified in the cause of humanity, we cannot understand how the altered financial position of the ministry could lessen the need for improvements in obstetrical practice. The matter, however, is closed. The Trustees of the Edward Wilson (*The Argus*) Trust withdrew this conditional offer and they together with others have taken their opportunities of contradicting in their own way rumours that were current. The Council of the University has now determined to create a chair of obstetrics. The vacancy was advertised in England many weeks ago and the advertisement is now appearing in our advertisement columns. Professor R. J. A. Berry assures us that the advisory committee is in no sense a selection committee. It is most reassuring to learn that the appointment of the new professor will be made in Melbourne on the records of each candidate without any guidance or recommendations from the committee in Great Britain.

The new professor will be required to devote the whole of his time to the work of his department. He is expected to give instruction, to conduct examinations, to supervise the work of the students, to undertake or organize research and possibly to act as State Director of Obstetrics. He is to be available for consultation with the medical officers of the Women's Hospital and of the Queen Victoria Hospital.

It is specifically set out that the professor will be responsible for the whole of the work in gynæcology and obstetrics, but not necessarily for the lecturing in gynæcology. This implies that he will be required to lecture on obstetrics. The opinion has been expressed in these columns on more than one occasion that it is unwise to require a professor to deliver systematic lectures. He should be the organizer of his department and should be freed from routine work as much as possible, in order that he may be able to coordinate the work of his staff and to expend his gifts and energies in guiding and controlling the teaching. The systematic lecture is a slow, unprofitable and unsatisfactory method of imparting knowledge. The professor could no doubt deliver occasional lectures with great advantage to the students, provided that he avoided the orthodox method of relating matters that are set out fully in every good text book. Clinical and informal lectures and demonstrations are admirable means of teaching. The student learns lessons from them that will be of value to him in his future practice. This is far more important than the acquisition of examination knowledge.

If the Faculty of Medicine and the Council of the University are prepared to give the professor a free hand to develope his department on sound lines, the plan sketched in the memorandum should serve as an ample and an excellent guide for a competent man. In particular it is highly advantageous to require the professor to devote the whole of his time to his duties and to include research among these duties. The salary of £2,000 a year is not a very generous one for a highly trained and practised obstetrician. In private practice he would make much more. It is, nevertheless, sufficient to attract a man with experience, high ideals and ability to work. It is announced that the appointment will be made about the beginning of July, but that the Council reserves to itself the right to fill the chair by invitation at any stage. If the Council chooses the right man either by invitation or by selection, the University of Melbourne should become one of the best training schools of obstetrics in the world.

Current Comment.

EOSINOPHILE LEUCOCYTES AND LEUCHÆMIA.

The difference between leucocytosis and myeloid leuchæmia is that in the latter condition elements which are not normally present, are introduced into the blood stream in large numbers. In addition to the polynuclear cells there are found their precursors, mononucleated granulated leucocytes. In the white cells all three types of granules are encountered, the neutrophile, the eosinophile and the basophile or mast cell granules. Dwarf forms of white blood corpuscles and mitotic figures are seen and nucleated red cells are always present. Each of these is of importance. During the last few years there have been reported several cases in which the leucocytes in the blood were of the adult and not of the immature type. In most instances the predominant cell has been the eosinophile cell. John Hay and William H. Evans have recently made a report in which they mention these cases and give details of two of their own.[1] The findings in each of the latter were essentially different and it is necessary that a short *résumé* of each should be given. The condition of the first patient was an unusual form of leuchæmia originating in the eosinophile system and was in every way comparable to acute myelogenous leuchæmia. The patient, aged forty-one years, was taken acutely ill three weeks before his death. The tonsils were enlarged and ulcerated, the inguinal lymphatic glands and the spleen were enlarged. Leucocytosis with gross eosinophilia was noticed at the commencement of the illness, but a complete blood examination was not made until the day before death. The erythrocytes numbered 3,975,000 per cubic millimetre, the hæmoglobin value was 60%. The white cells numbered 72,187 per cubic millimetre. Polymorphonuclear neutrophile cells and neutrophile myelocytes numbered 4·6%, lymphocytes 6%, mononuclear cells 1·4%. Basophile cells were 4·3% and eosinophile cells 83·7%. Of the latter 75·5% were polymorphous, 3·8% were myelocytes and 4·4% metamyelocytes (cells with nuclei which were simply indented and not divided into lobes). A limited *post mortem* examination was possible. Only one piece of bone (part of a rib) was removed. The bone marrow was greyish-white and almost entirely cellular; there was an excess of leucoblastic tissue in which eosinophile cells were very prominent, myelocyte and polynuclear forms being practically equal. Small numbers of neutrophile and basophile myelocytes and polynuclear cells were seen and there were numerous non-granular cells, apparently myeloblasts. In the spleen the lymphoid follicles were almost completely obscured and infiltrated by eosinophile cells; the pulp was also crowded with these cells and the sinuses contained large numbers of them. Phagocytosis of eosinophile cells by mononuclear cells of the pulp and by the endothelial cells lining the sinuses could be seen. Hay and Evans hold that the appearances did not afford any evidence of active formation of eosinophile cells in the spleen, they were apparently due to an infiltration from the blood stream. None of the organs showed any evidence of tuberculosis or of any other lesion which would account for the eosinophilia. During the life of the patient the stools were examined for parasites, protozoa and ova, but without result.

In the authors' second case the condition ran a much more chronic course. It was regarded as polycythæmia with eosinophilia, eosinophilic erythroleuchæmia. The patient, a woman, aged fifty-four years, was under observation for eight months before her death. She had complained of vague symptoms for two years before that time. The erythrocytes in August, 1925, numbered 7,450,000 per cubic millimetre; the number subsequently rose to over 8,000,000 and at the time of death was 7,000,000. The white cells in August 1925 numbered 62,180 per cubic millimetre. They fell in number after this, on one occasion the number was 16,250 and at the time of death was 26,562. The percentage of eosinophile cells in August, 1925, was 16·1; it subsequently rose to 55·2 and at the time of death was 35·6. On *post mortem* examination the bone marrow was found to be entirely cellular. The cellular tissue between the capillaries manifested an almost equal division into erythroblastic and leucoblastic elements. In the spleen there were found vascularity, fibrosis, atrophy of lymphoid structures and diffuse eosinophile infiltration.

[1] *The Quarterly Journal of Medicine*, January, 1929.

It will be seen that these two cases presented features of unusual interest. The conclusions of Hay and Evans are of a general nature. They state that they are convinced that there is a category of cases indistinguishable from myeloid leuchæmia except for the presence of selective disturbance of individual mature elements. They draw particular attention to the fact that the condition of their first patient was acute and that the eosinophile cells were no more immature than the other blood cells. They agree with McDonald and Shaw that it is idle to attempt to draw a distinction between an eosinophile leucocytosis in response to a definite toxic influence and a true leuchæmic condition. The underlying cause of leuchæmia is still unsettled and while it seems reasonable to describe it as a neoplasm of the hæmopoietic system, they point out that the ultimate stimulus to neoplastic growth in the bone marrow is just as obscure as in any other body tissue. As far as the second case reported is concerned, Hay and Evans enumerate the links between *polycythæmia vera* and myelogenous leuchæmia and use them as an argument in favour of the dualistic view of the origin of the blood cells. This aspect of the question need not be considered at present. It will be of more interest to discuss the significance of the eosinophile increase and its possible relationship to the cellular increases in the blood.

The first fact to be remembered is that the neutrophile, eosinophile and basophile cells are absolutely differentiated from one another by the nature of their protoplasm. This is evidenced not only by the different types of granulation, but by their chemotactic susceptibility to stimulation. Substances which call forth either a positive or negative chemotaxis in one group of cells, fail to exert any influence on another group. Sometimes the opposite effect is noted and a substance produces an attraction for one kind of cell and a repulsion for another kind. Further, Ehrlich showed that one kind of cell is the carrier of one kind of granule only. He also taught that the granules are the secretion products of the cells. He was led to this view by the observation that in pernicious anæmia the polynuclear cells of the blood and of the bone marrow and their precursors are free from neutrophile granulation. This view was in direct opposition to that held by Altmann that the granules are the elements which supply the cell with oxygen. Ehrlich pointed out that, if this were true, disappearance of granulation would lead to death of the cell. It is thus clear that eosinophilia may be regarded as the response of the certain type of leucocyte to a specific stimulus. Just as the neutrophile cells respond to the stimulus provided by toxins of the organisms of suppuration, so eosinophile cells respond to such conditions as asthma, pemphigus, scarlatina, helminthiasis *et cetera*. It should be pointed out here that the origin of eosinophile cells from the bone marrow is accepted and that the presence of eosinophile cell infiltrations around trichinæ and the peribronchial infiltrations in asthma are the result of chemotaxis. If it is idle to draw a distinction between an eosinophilic leucocytosis and a true leuchæmia, the whole matter is reduced to a consideration of the nature of the stimulus and of the state of the bone marrow. There is no reason why one of these factors should be considered to the exclusion of the other. It is impossible in considering the first case of Hay and Evans to determine the nature of the stimulus. It is possible that if a stimulus of sufficient magnitude had been in operation, a bone marrow inherently weak might continue a response which would have been of short duration, had the bone marrow been normal. It should be pointed out that in the first case of Hay and Evans none of the usual causes of eosinophilia was found. The *post mortem* examination, however, was a limited one and it would be quite possible for an undiscovered focus to have been present. It must also be remembered that it is not likely that all the causes of eosinophilia have been discovered. In the second case of these authors any stimulus which was present (there may have been more than one stimulus) must have acted on both the apparatus controlling the production of eosinophile cells and the erythropoietic apparatus. Research in the future must be directed to the eosinophile cell and to the stimuli which act upon it. Some work has recently been carried out by Gregory Schwartzman on the effect of tuberculin on normal leucocytes *in vitro*. He found that tuberculin possesses *in vitro* a toxicity for normal leucocytes and that tuberculin is able to influence the morphological appearances of the monocytes to a large extent. The cells were altered in shape and sometimes formed groups. From these groups giant multinucleated cells originated. It is possible that some work of this nature on eosinophile cells would yield useful information.

HEMIPLEGIA OCCURRING AFTER DIPHTHERIA.

Although paralysis is commonly seen after diphtheria, the occurrence of hemiplegia in this regard is a rare event. D. W. Winnicott recently reported to the Royal Soceity of Medicine hemiplegia in a child of two years and five months.[1] The child suffered from nasal diphtheria and the paresis appeared while it was in hospital. J. D. Rolleston in discussing the case pointed out that hemiplegia was usually the result of embolus from cardiac thrombosis and emboli were found in other parts than the brain. He doubted whether the hemiplegic symptoms in the patient were due to diphtheria. In his experience purely nasal diphtheria did not tend to affect the heart in the same way that faucial diphtheria did. At the same time he offered no suggestions as to the cause in this instance. The history is so meagre that no conclusion can be formed. It would be interesting to know why nasal diphtheria does not involve the heart.

[1] *Proceedings of the Royal Society of Medicine*, February, 1929.

Abstracts from Current Medical Literature.

SURGERY.

Animal Blood Injections for Tuberculosis.

E. Kisch and E. Bergmann (*Münchener Medizinische Wochenschrift*, October 19, 1928) have employed intravenous injections of animal blood in the general treatment of severe tuberculous lesions suitable for surgical treatment and complicated by mixed infection as well as in empyema and pulmonary tuberculosis associated with abdominal lesions. Injections of citrated blood from sheep, cattle, horses and pigs were given at intervals of a week. It is important to allow ample time during the injections and the author recommends five minutes per cubic centimetre. Rigors, headache and vomiting are common, also a temporary albuminuria. A localized urticaria around the site of injection is also commonly seen. He considers that before the course is commenced, a preliminary injection of two cubic centimetres of sheep's blood should be given slowly and if no reaction occurs, the larger doses employed (three to five cubic centimetres) can be continued one to four days later. Patients yielding a definite reaction received most benefit. If an increase in weight and improved appetite did not follow a course, two further injections of pig's blood were given usually with good results. In all forty-two patients have been treated. Ten received only one injection and further treatment was abandoned because of the poor general condition. Of the remaining thirty-two in only four was no improvement noted. Unlike the conditions seen in pulmonary tuberculosis the gain in weight did not run parallel with improvement in the tuberculous lesion. The lesions were all advanced and complicated with mixed infections and sanatorium treatment alone had no effect on them. It required the stimulus of the foreign protein to do this and the authors with the approval of Bier consider that the use of animal blood is justified by the results they have obtained.

Injection Treatment of Varicose Veins.

E. Vogt (*Monatsschrift für Geburtshülfe und Gynäkologie*, December, 1928) recommends the use of injections of sodium chloride solution to produce thrombosis in varicosities of the lower extremities. He considers that the results of the injections have more than a local effect on the enlarged vessels, since apart from this, the blood pressure is raised and the heart becomes stronger in its action. Attempts were also made to treat varicose veins during pregnancy, but were not very successful. In one patient with extensive varicosities of the vulva for whom induction of abortion was being considered, a portion of the mass was injected with immediate and lasting relief. No untoward results, especially embolism, were noted in the series of cases which he has reported.

Osseous Changes in Congenital Syphilis.

S. Engel and S. Schmidt (*Klinische Wochenschrift*, October 28, 1928) have systematically examined the limbs of infants suffering from congenital syphilis and state that bony changes occur invariably in all such children. Osteochondritis is the most prevalent condition present and next to it periostitis. Osteochondritis is observed mainly in the distal ends of the extremities—75% in the forearm and leg as compared with 25% in the upper arm and thigh. The incidence of periostitis is similar, but it is noteworthy that if only one bone be affected, it is always the tibia. The authors consider that the predilection of syphilis for the distal portions of both limbs is largely due to the more constant movements of the legs and forearms of the infant as compared with the thighs and upper arms. The tibia is also under some muscular strain long before walking and this is confirmed by the frequency of bowing of the legs in normal infants. They conclude, therefore, that the general disposition of the infantile skeleton to congenital lesions is connected with the mechanical movements of early life. The forearm is the most important limb to inspect for any syphilitic lesions.

Bony Carcinomatous Metastases.

R. Kienböck (*Wiener Medizinische Wochenschrift*, October 27, 1928) states that in the presence of bony carcinomatous metastases often no primary tumour can be discovered. Sometimes while a primary focus is obvious, there is gross discrepancy in size between it and the secondary growths. The latter may be single or multiple, hard or soft, painful or painless, while deformation of bony structure may not be observed. Spontaneous fracture may be the first symptom. Some patients appear to be in good health with no signs of cachexia and the most puzzling diagnostic problems often occur in young people. From the radiological point of view he divides metastatic growths into two groups—the osteolytic with destruction of bone and the osteopoietic associated with the formation of sclerosed bone. The first type is generally associated with a central area of rarefaction which contrasts clearly with the normal darker shadow of healthy bone. This distinction is not so pronounced in chronic bone metastases nor in elderly persons. Diffuse infiltration is noted in the second group. On the whole if the primary growth be a soft one, metastases belong to the osteolytic group, while primary tumours such as scirrhus of the breast are associated with osteopoietic growths. The cause of localized pain and weakness can often be diagnosed only after radiological examination. This shows that it is due to a subperiosteal fracture or fissure and it is a frequent sequela of the first group. He next refers to the differential diagnosis of metastases and myelomata, *osteitis fibrosa* and *osteitis deformans*. Treatment largely depends on accurate and early diagnosis. He considers that in cases of solitary tumour formation, provided that there are no other growths in the lungs or abdominal viscera and that the patient is in good general condition, resection at the most is indicated. Naturally, careful search must be made for the primary focus which is removed if possible. As in many instances this is not possible, treatment of both primary and secondary growths is limited to radiotherapy. He concludes with the warning that accurate diagnosis of bony growths is difficult and one only for the expert radiologist.

Syphilis of the Stomach.

H. A. Singer and K. A. Meyer (*Surgery, Gynecology and Obstetrics*, January, 1929) report in detail two cases of syphilis of the stomach and discuss the subject. They have studied the condition in four patients subjected to gastric resection. At the same time they have made a careful search for syphilitic lesions of the stomach in their autopsy material. No example was found among five thousand bodies, although 10% of them manifested extragastric signs of the disease. They have concluded that syphilis of the stomach in a microscopically recognizable form appears more frequently at the surgical than at the autopsy table. As far as they can judge from the literature this is a universal experience. They regard the present situation relating to the incidence of syphilis of the stomach as analogous to the former status of duodenal ulcer and point out that it was only after repeated surgical demonstration that pathologists and clinicians subscribed to the idea that duodenal ulcer was far more common than former autopsy statistics indicated. They conclude by analogy with duodenal ulcer that retrogression of the syphilitic infection in the stomach frequently occurs. Many of the lesions diagnosed at autopsy as benign pyloric hypertrophy, hour-glass stomach and *linitis plastica*, represent gastric syphilis encountered in the healing or healed stage. In support of the view that syphilis of the stomach tends to heal and in healing to lose its characteristic features, the authors point out that it is possible to identify in a series of lesions what are apparently transitions between the active and the healed stages of the infection. Even in a single specimen different stages of the inflammatory reaction may be encountered. In the two patients reported by the authors no spirochætes were found in the gastric specimens. Positive responses to the Wassermann test were obtained, however. In one instance hepatic gummata were found and in both the radiological findings, the achylia and the microscopical appearances were regarded as sufficient to confirm the diagnosis.

Ulcer Cruris.

H. O. McPHEETERS (*Surgery, Gynecology and Obstetrics*, October, 1928) describes the ætiology, pathogenesis and treatment of *ulcer cruris*. This is the commonly known varicose ulcer of the leg. It is a trophoneurotic disturbance and condition resulting secondarily to the development of varicose veins. The ulcer is the end result of the stagnation of blood serum in the tissues secondary to the veins. The attempt to cure the ulcer first and the veins second is wrong both in theory and practice. The varicose veins are obliterated far better by injection treatment than by operation. The supportive bandage for the affected extremity with the rubber sponge pressure over the ulceration is the oldest and yet the most efficient treatment of the present day. Judicious employment of the skin graft at the proper time greatly shortens the period of healing. All the long standing ulcers with extensive involvement must have continued support for long periods of time if recurrence is to be avoided. The duration and extent of the support must be decided in each individual case. Finally, by the use of the described technique all varicose ulcers can be healed and kept healed. If they cannot, it means that the operator has not located the vein which causes the condition and which is often under the ulcer bed, or that he has been negligent in giving the extremity the necessary support.

Inguinal Hernorrhaphy.

E. M. HODGKINS (*Surgery, Gynecology and Obstetrics*, December, 1928) describes a new method of inguinal herniorrhaphy with living fascial sutures obtained from the rectus sheath. For many years there has been a feeling of dissatisfaction among surgeons with the method of using catgut sutures under tension in muscle tissue. Kirschner's free transplant of living *fascia lata* to reinforce the suture line of the external oblique aponeurosis was undoubtedly a step forward in fascial surgery. Koontz advocated ox tendon and fascia, an experiment receiving much favourable comment. Gallie and Le Mesurier have made the most important contribution with the recommendation that "living sutures" of *fascia lata* be used in place of catgut. There is an increased liability to wound infection, but nevertheless the method is surgically sound. The three fundamental principles at the present time are high and firm ligation of the sac, closure of the defect in the abdominal wall by suture of the muscle, aponeurosis or both to the inguinal ligament and the narrowing of the external ring. All three must be done to make the operation complete. The writer has evolved a living suture technique by fascia weaving. A skin incision is made to give adequate exposure of the inguinal canal and the whole width of the rectus sheath. The canal is opened in the usual manner except that the external oblique aponeurosis should not be dissected off the internal oblique, as the natural adhesion is a source of strength. The cut-off neck of the sac is fixed by a modified Kocher's technique to the under surface of the external oblique through an aperture made in the internal oblique muscle. Parallel strips of anterior rectus sheath are cut horizontally with an attached lateral margin. The strips are eight millimetres (five-sixteenths of an inch) apart and are the whole width of the rectus sheath. About four strips are sufficient. The strip is threaded into the eye of a needle and tied there with catgut. The fascial strip is drawn through the medial leaf of the external oblique muscle as close as possible to the line of surgical separation of the aponeurosis and the internal oblique. The edge of the internal oblique muscle is pierced from above downwards and the suture is drawn through to the under side. The strip is passed through the shelving portion of the inguinal ligament in the usual manner from below upwards and is drawn taut. The edge of the internal oblique muscle is again pierced from above downward and is picked up just above the first point of suture. Finally, the needle is brought up through the fascial strip itself. The strip is drawn through and anchored securely at this point with a single tie of linen or number 0 chromicized catgut. Fascial weaving does not cause muscle necrosis. The fascial sutures are surely living sutures.

The Pathology of Epididymitis.

H. C. ROLNICK (*Surgery, Gynecology and Obstetrics*, December, 1928) describes his conclusions resulting from experiments on the mode of infection and spread and the pathology of acute epididymitis in the first few days. Epididymitis does not follow vasotomy except in those cases in which there has been a secondary infection of the vasotomy wound. Acute epididymitis at its onset consists of an interstitial and peritubular and not an intratubular inflammation of the tail as well as the body and head of the epididymis. The extension of the infection from the tail is *viâ* the peritubular and interstitial and not the intratubular tissues. Direct medication to the *vas deferens* does not influence the infectious progress, because the bacteria are in the interstitial tissues of the walls and not in the lumen. Occlusions of the epididymis results in the main from interstitial and peritubular inflammation producing compression and destruction from without in acute epididymitis and fibrosis and scarring from without in chronic epididymitis. Epididymotomy does not consist of nicking or incising the dilated tubules of the epididymis presumably filled with pus, because the inflammation is interstitial. Early relief of the tension is definitely indicated not only to relieve the symptoms, but to prevent destruction and obliteration of the tubules. It should be limited to the tail of the epididymis without incision of the *tunica vaginalis*. The purpose is to relieve tension and provide drainage from the interstitial tissues. Epididymotomy should consist of one clean incision. Relief of tension will reduce the incidence of occlusion and resultant sterility. The operation should be performed early within the first few days after the onset of an epididymitis.

Treatment of Injuries Caused by Electricity.

S. JELLINEK (*Wiener Medizinische Wochenschrift*, January 19, 1929) considers that sufficient attention has not been given to the treatment of injuries caused by electrical currents. Too frequently patients recover from the trauma of the initial shock and burns only to succumb to the trauma of the surgical knife. It is not generally recognized that there is a latent period lasting days or weeks during which necrosis of bones and soft parts may occur. Therefore he appeals for conservatism in treatment. The main contraindication to immediate operation is the risk of necrosis of the connective tissues during the first week as well as the likelihood of thrombosis or hæmorrhage as a result of structural alterations in vessel walls. However, immediate surgical interference is indicated in the following instances. Lumbar puncture is an important life-saving procedure when symptoms of increased cerebral pressure are present. The acute œdema of the brain causes definite raising of the pressure. In many instances this may be delayed for some hours. Complaint is made of headache, giddiness and weakness, the pulse is irregular and signs of mental irritation develope which may pass on to fits. The removal of twenty to thirty cubic centimetres of fluid leads to rapid improvement. The next indication for surgical intervention is secondary hæmorrhage. The vascular system forms an ideal conductor for the electrical current and is generally damaged at various points. During the third and fourth weeks in particular there is grave risk of secondary hæmorrhage from destruction of arteries, especially those in necrotic areas. Therefore, tourniquets must always be kept at hand and the nursing staff warned of the possibilities. The third indication is amputation of a limb owing to extensive damage associated with severe general reaction. Naturally this is a rare complication and operation is only to be performed as an urgent life-saving measure after the failure of other treatment. The final indication for operation is the primary suture of severed nerves especially those of the hand. While conservatism is generally indicated, it does not mean a policy of *laissez aller*. It implies careful treatment of the local lesions by foments, baths *et cetera*, X ray examination of bones and joints, the investigation of possible nerve lesions, constant examination of the urine and careful nursing.

British Medical Association News.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, 30-34, Elizabeth Street, Sydney, on March 21, 1929, DR. J. E. V. BARLING, the President, in the chair.

ANNUAL REPORT OF THE COUNCIL.

The Honorary Secretary presented the annual report of the Council and moved that it be received. The motion was seconded by DR. A. DAVIDSON and carried. The report is as follows:

THE Council presents the following report on the work of the Branch for the year ended March 21, 1929.

Membership.

The membership of the Branch is now 1,699, as compared with 1,661 at the date of the last report, showing a net increase of 38.

The additions have included elections and resumptions of membership, 80; removals into the area of the Branch, 41. The losses have included resignations, 5; removals out of the area of the Branch, 40; default in payment of subscription, 21; deaths, 17.

The losses by death have been: Dr. J. P. Fitzsimmons, Dr. E. E. Griffiths, Dr. T. Morgan Martin, Dr. R. Roger, Dr. J. Oswald, Dr. J. B. Wilson, Dr. F. L. Bignell, Dr. R. S. Bowker, Dr. W. T. J. Newton, Dr. E. H. Florance, Dr. D. Clark, Dr. S. H. MacCulloch, Dr. J. J. Brennan, Dr. H. J Clayton, Dr. W. J. O. Walker, Dr. N. J. Dunlop, Dr. F. T. Allen.

Meetings.

Ten ordinary meetings of the Branch, including the annual meeting, one extraordinary meeting, six clinical meetings and one general (scientific) meeting, were held. The average attendance was 59. The ordinary meetings, as follows, were held in conjunction with meetings of sections, namely: April 26, with the Section of Surgery and the Section of Radiology; May 31, with the Section of Medicine and the Section of Neurology and Psychiatry; June 28, with the Section of Obstetrics and Gynæcology; July 26, with the Section of Medical Literature and History; September 27, with the Section of Hygiene and Preventive Medicine and the Section of Pathology and Bacteriology; October 25, with the Section of Orthopædics, the Section of Surgery and the Section of Pædiatrics; November 29, with the Section of Hygiene and Preventive Medicine and the Section of Medicine.

The extraordinary meeting of September 27 was concerned with the appointment of honorary medical staff at the Royal Prince Alfred Hospital.

The clinical meetings were held at the Education Department's Special School at Glenfield, the Royal Prince Alfred Hospital, the Sydney Hospital, the Royal North Shore Hospital, the Royal Alexandra Hospital for Children and Saint Vincent's Hospital.

The business of the meetings during the year included twenty-three papers and addresses and numerous reports of cases and exhibits and lantern demonstrations.

Representatives.

The Branch was represented as follows:

(*a*) *Council of the British Medical Association* (1928-1929): Sir T. Jenner Verrall, LL.D., Vice-President of the British Medical Association.

(*b*) *Representative Body* (1928-1929): Representative, Dr. C. B. Blackburn, O.B.E.; Deputy-Representative, Dr. F. Brown Craig.

(*c*) *Federal Committee of the British Medical Association in Australia*: (1928) Dr. J. A. Dick, C.M.G., Dr. R. H. Todd; (1929) Dr. J. A. Dick, C.M.G., Dr. R. H. Todd.

(*d*) *Australasian Medical Publishing Company, Limited*: Dr. George Armstrong, Dr. T. W. Lipscomb, Dr. F. P. Sandes.

(*e*) *Council of the Bush Nursing Association* (1928-1929): The President, Dr. J. E. V. Barling.

(*f*) *Council of the Royal Society for the Welfare of Mothers and Babies*: Dr. R. B. Wade, Professor J. C. Windeyer.

(*g*) *Board to Control Campaign Against Tuberculosis*: Dr. S. A. Smith.

Council.

(*a*) The attendance of the members of the Council and of the standing committees was as set out in the table on the next page.

(*b*) The representatives of the Local Associations of Members, appointed on the invitation of the Council to attend the regular quarterly meetings of the Council, were as follows: Dr. W. Brodie Grant (Balmain District), Dr. J. M. Alcorn (Central Southern), Dr. K. S. M. Brown (Central Western), Dr. A. M. Gledden (City), Dr. H. Hunter (Eastern Suburbs), Dr. W. F. Simmons (Illawarra Suburbs), Dr. W. E. Kay (Northern District), Dr. Harold Norrie (Northern Suburbs), Dr. N. M. A. Alexander (South Sydney), Dr. J. Brooke Moore (Western), Dr. R. J. Nixon (Western Suburbs).

Library.

Dr. J. A. Dick was again appointed to the position of Honorary Librarian. Donations of books and periodicals were received from the Australasian Medical Publishing Company, Limited, the late Dr. N. J. Dunlop, Dr. W. H. Crago, Dr. F. Barrington, Dr. W. Chisholm and others. Gifts of interesting and valuable prints and photographs have been made by Dr. C. B. Blackburn, Dr. F. Barrington, Dr. E. M. Humphery and Dr. W. Chisholm, who also presented a Lister Carbolic Spray. The *British Journal of Surgery*, the *Annals of Surgery*, the *Journal of Bone and Joint Surgery* and the *Medical Press and Circular* have been added to the current periodicals available.

Affiliated Local Associations of Members.

The following is a list of the Local Associations of members and their Honorary Secretaries:

Balmain District: Dr. R. K. Burnett (Rozelle).

Border: Dr. R. Affleck Robertson (Albury).

City: Dr. H. A. Ridler (159, Macquarie Street, Sydney).

Central Northern: Dr. A. T. Roberts (Newcastle).

Central Southern: Dr. R. G. Woods (Goulburn).

Central Western: Dr. K. S. Macarthur Brown (Parramatta).

Eastern Suburbs: Dr. Hugh Hunter (Waverley).

Eastern District: Dr. A. Muscio (Taree).

Illawarra Suburbs: Dr. W. F. Simmons (Bexley).

Northern District: Dr. R. J. Jackson (Armidale).

North Eastern: Dr. A. J. Opie (Lismore).

Northern Suburbs: Dr. E. M. Humphery (Killara).

South Eastern: Dr. H. H. Lee (Wollongong).

South Sydney: Dr. J. G. Hunter (Mascot).

Western: Dr. S. R. Dawes (Orange).

Southern District: Dr. C. R. Sim (Wagga).

Western Suburbs: Dr. J. F. Walton (Summer Hill); Dr. W. M. A. Fletcher (Haberfield), Assistant Secretary.

Annual Meeting of Delegates.

The sixteenth annual meeting of the delegates of the affiliated Local Associations of members with the Council was held on October 5, 1928, at the B.M.A. Library, Sydney. An account of the meeting appeared in THE MEDICAL JOURNAL OF AUSTRALIA of November 3, 1928, at page 568, and a report of the proceedings was sent to the several Local Associations. The delegates present at the meeting were as follows: Dr. W. B. Grant (Balmain District), Dr. C. A. F. Clark (Central Northern), Dr. G. A. Buchanan (Central Southern), Dr. K. S. M. Brown (Central Western), Dr. A. M. Gledden (City), Dr. S. A. Railton (Eastern District), Dr. H. Hunter (Eastern Suburbs), Dr. W. F. Simmons (Illawarra Suburbs), Dr. A. G. Brydon (Northern District), Dr. O. A. Diethelm (North Eastern), Dr. H. F. J. Norrie (Northern Suburbs), Dr. A. M. Davidson

(South Sydney), Dr. J. T. Paton (Western), Dr. A. M. McIntosh (Western Suburbs).

Sections for Special Branches of Medical Knowledge.

(*a*) Pædiatrics (inaugurated October 4, 1921): *Chairman*, Dr. E. H. M. Stephen; *Honorary Secretaries*, Dr. F. C. Rogers and Dr. M. J. Plomley. Membership, forty-two. Meetings were held on March 23, June 22, July 27, August 24, October 25 (in conjunction with the Branch, the Section of Surgery and the Section of Orthopædics).

(*b*) Hygiene and Preventive Medicine (inaugurated January 3, 1922): *Chairman*, Dr. R. Dick; *Honorary Secretary*, Dr. E. S. Morris. Membership, twelve. Meetings were held on September 27 (in conjunction with the Branch and the Section of Pathology and Bacteriology) and November 29 (in conjunction with the Branch and the Section of Medicine).

(*c*) Orthopædics (inaugurated May 7, 1923): *Chairman*, Dr. C. Nigel Smith; *Vice-Chairman*, Dr. H. R. G. Poate; *Honorary Secretary*, Dr. J. Hoets. Membership, eighteen. Meetings were held on April 19, June 21, August 23 and October 25 (in conjunction with the Branch and the Section of Surgery and the Section of Pædiatrics).

(*d*) Pathology and Bacteriology (inaugurated April 3, 1924): *Chairman*, Dr. W. K. Inglis; *Honorary Secretary*, Dr. F. S. Hansman. Membership, twenty-two. Meetings were held on March 21, August 23, September 27 (in conjunction with the Branch and the Section of Hygiene and Preventive Medicine), September 28, November 22.

(*e*) Neurology and Psychiatry (inaugurated June 5, 1924): *Chairman*, Dr. A. Davidson; *Vice-Chairman*, Dr. N. D. Royle; *Honorary Secretary*, Dr. J. A. L. Wallace; *Honorary Medical Secretary*, Dr. R. A. Noble. Membership, forty-eight. Meetings were held on May 31 (in conjunction with the Branch and the Section of Medicine), November 8 and December 7.

(*f*) Oto-Rhino-Laryngology (inaugurated June 11, 1924): *Chairman*, Dr. H. J. Marks; *Honorary Secretary*, Dr. Garnet Halloran. Membership, twenty-six. Meetings were held on July 24, October 18, December 4.

(*g*) Medicine (inaugurated October 1, 1924): *Chairman*, Dr. J. Macdonald Gill; *Honorary Secretary*, Dr. Cotter Harvey. Membership, thirty. Meetings were held on May 31 (in conjunction with the Branch and the Section of Neurology and Psychiatry), November 29 (in conjunction with the Branch and the Section of Hygiene and Preventive Medicine), December 20.

(*h*) Medical Literature and History (inaugurated June 26, 1925): *Chairman*, Dr. R. Scot Skirving; *Honorary Secretary*, Dr. L. Cowlishaw. Membership, twenty-eight. Meetings were held on April 24, July 26 (in conjunction with the Branch), September 29, November 1.

(*i*) Surgery (inaugurated July 30, 1925): *Chairman*, Dr. C. E. Corlette; *Honorary Secretary*, Dr. T. Farranridge. Membership, twenty seven. Meetings were held on February 21, April 26 (in conjunction with the Branch and the Section of Radiology), July 4, October 25 (in conjunction with the Branch and the Section of Orthopædics and the Section of Pædiatrics), December 5.

(*j*) Obstetrics and Gynæcology (inaugurated August 6, 1925): *Chairman*, Dr. R. L. Davies; *Vice-Chairman*, Dr. J. L. T. Isbister; *Honorary Secretary*, Dr. A. J. Gibson. Membership, forty-nine. Meetings were held on March 21, June 28 (in conjunction with the Branch), September 19, November 21.

(*k*) Radiology (inaugurated December 3, 1926): *Chairman*, Dr. J. G. Edwards; *Honorary Secretary*, Dr. M. Frizell. Membership, nineteen. Meetings were held on April 26 (in conjunction with the Branch and the Section of Surgery), November 2.

(*l*) Genito-Urinary and Venereal Diseases (inaugurated August 7, 1928): *Honorary Secretary*, Dr. P. Fiaschi.

(*m*) For the Study of Cancer (inaugurated October 10, 1928): *Chairman*, Dr. H. G. Chapman; *Honorary Secretary*, Dr. H. M. Moran. Membership, sixteen. Meetings were held on November 15, 1928, and March 14, 1929.

Federal Committee.

The Federal Committee of the British Medical Association in Australia met in Melbourne on April 4, 1928, and

ATTENDANCE OF MEMBERS OF THE COUNCIL AND OF THE STANDING COMMITTEES OF THE COUNCIL.

Office-Bearers.	Council.	Executive and Finance Committee.	Ethics Committee.	Organization and Science Committee.	Medical Politics Committee.	Medical Journal Sub-Committee (Executive and Finance Committee).
DR. A. J. ASPINALL	8	—	—	4	—	—
DR. J. E. V. BARLING (President)	10	11	4	8	11	10
DR. GEORGE BELL, O.B.E.	11	14	—	—	—	—
DR. C. B. BLACKBURN, O.B.E.[1]	3	—	1	—	—	—
DR. F. BROWN CRAIG (President Elect)[2]	6	11	1	4	5	—
DR. W. H. CRAGO (Honorary Treasurer and Premises Attorney)	11	18	5	11	10	9
DR. A. DAVIDSON	10	—	5	—	—	—
DR. J. A. DICK, C.M.G. (Honorary Librarian)	10	19	—	—	11	—
DR. J. GOODWIN HILL	8	—	—	—	9	—
DR. A. W. HOLMES À COURT	7	—	—	8	—	—
DR. E. M. HUMPHREY	7	10	—	—	7	—
DR. J. L. T. ISBISTER	10	—	5	—	—	—
DR. C. H. E. LAWES	8	6	—	—	11	—
DR. T. W. LIPSCOMB[3]	8	13	—	—	9	5
DR. R. J. MILLARD, C.M.G.	8	14	—	—	—	5
PROFESSOR A. E. MILLS	5	—	—	6	—	—
DR. A. A. PALMER	9	—	5	—	—	—
DR. S. A. SMITH (Past President)	6	11	—	—	—	—
DR. R. H. TODD (Honorary Secretary)	10	18	5	10	12	10
DR. R. B. WADE	8	16	4	—	—	10
Number of Meetings held	11	20	5	11	12	11

[1] Absent on leave February to October, 1928. [2] Absent on leave February to October, 1928. [3] Absent on leave March to June, 1928.

in Sydney on August 7, 1928. Reports of the proceedings of the Committee will be found in THE MEDICAL JOURNAL OF AUSTRALIA of May 5, 1928, at page 562, and of August 25, 1928, at page 249, respectively.

Australasian Medical Congress (British Medical Association).

Arrangements have been made for the third session of Congress, which is to be in Sydney, to be held at the University of Sydney, Monday, September 2, to Saturday, September 7, 1929. Sir Alexander MacCormick, who had graciously accepted appointment as President, has been obliged, unfortunately, to relinquish the position owing to the necessity having arisen for him to be away from Australia at the time. Dr. G. H. Abbott has allowed himself to be nominated in his place. Organization of the session is well advanced and much interest is being taken throughout all the States in the preparatory work of the sectional meetings. Attention is drawn to the constitution and regulations of Congress which provide for every member of any Branch of the British Medical Association in Australia and of the New Zealand Branch to become a member of Congress for the session upon his own application for the purpose and without election upon payment to the Executive Committee of the prescribed subscription. The Honorary General Secretaries, Dr. A. A. Palmer and Dr. T. W. Lipscomb, and the Honorary Treasurer, Dr. W. H. Crago, have been continuously engaged in the work of organization.

Congress is to be honoured by the presence of the President of the British Medical Association, Sir Ewen Maclean, M.D., F.R.C.P., F.R.S.E., who will visit Australia for the purpose on behalf of the Association.

"The Medical Journal of Australia."

THE MEDICAL JOURNAL OF AUSTRALIA has maintained its reputation as a medical journal of the highest scientific interest and the Printing House with its extended plant is becoming more recognized by the profession not only in New South Wales but in other States for supplying the ordinary daily requirements of practitioners.

Premises.

It is anticipated that the new premises of the Branch now being built in Macquarie Street, Sydney, will be ready for occupation in May, 1930. The architectural competition, which closed on March 8, 1928, resulted in the presentation of fifty-one designs received from forty-eight architects in Sydney, Melbourne and Brisbane. Many of them were of the highest merit and of great interest. The competition was judged by Professor Leslie Wilkinson and prizes were awarded, the first to Messrs. J. C. Fowell and K. McConnel, the second to Messrs. Peddle, Thorp and Walker, and the third to Mr. Keith McCredie and Miss D. Weatherstone. Mr. Fowell and Mr. McConnel were appointed the architects. Plans were adopted and specifications approved. Tenders for the building contract were invited. That of Messrs. Hutcherson Brothers was accepted, to commence December 1, 1928. The demolition of 135-137, Macquarie Street was proceeded with. The new building will consist of a basement, ground floor which will contain the Assembly Hall, first floor which will comprise the library, council and committee rooms and offices, the second to twelfth floors designed as consulting rooms. The second floor is being so constructed as to allow of its bearing the weight of a library in any part if required in time to come. The management of the new building has been put into the hands of Messrs. Hardie and Gorman, Estate Agents, Martin Place. Rentals have been fixed and a large number of the consulting rooms have been bespoken by members of the Branch and dentists, for whose accommodation special provision has been made in certain rooms on each floor.

In regard to the resumption of 30-34, Elizabeth Street by the Municipal Council of Sydney, the amount of compensation payable to the Branch still awaits determination by the Land Court.

The debenture issue authorized for the purpose of assisting the financing of the new building has not yet been all taken up. The issue consisted of 600 debentures (Series "B") of £50 each (£30,000), carrying interest at the rate of £6 *per centum per annum*.

Contract Attendance, Friendly Society Lodges.

The complication resulting from the passing of the *Workers' Compensation Act*, 1926-1927, of the relations of medical officers of friendly society lodges and their lodge patients who happened to be injured workers entitled to compensation (cost of treatment) from their employers under the Act, has been satisfactorily adjusted so far as those medical officers are concerned whose agreements with the lodges have been amended so as to meet the position by means of the "Supplemental Agreement" of April 1, 1928, duly entered into. All agreements entered into since that date are, of course, free from complication in that respect.

Workers' Compensation Act, 1926-27.

The arrangement of October 4, 1927, between the Government Insurance Office, the associated and certain other licensed insurers and the Council, under which the insurers who are parties to it undertake to pay directly to medical attendants of "injured workers" their fees and charges subject to these being in accordance with a schedule agreed upon (Schedule "D"), appears to have worked with some considerable measure of satisfaction to the members interested, as well as to the insurers concerned. A large number of inquiries, however, have been received both from medical attendants and insurance offices. They have been dealt with by the Medical Politics Committee, whose work during the year has in consequence been much increased. In regard to attendance on injured worker railway employees, although it was thought that the Act had made the Railway Commissioners liable to compensate their employees for the cost incurred by them in obtaining the necessary medical, surgical and hospital treatment, the determination given by the Workers Compensation Commission in The Treasurer, Royal Prince Alfred Hospital *versus* The Railway Commissioners of New South Wales, reported in the Workers' Compensation Reports, Volume II, page 123, is to the effect that if the injured worker employee is in receipt of full wages under the provisions of the *New South Wales Government Railways Act*, 1912, Section 100 A, he cannot recover compensation for the expenses incurred by him for his treatment.

Police Offences Amendment (Drugs) Act, 1927.

The provisions of this Act, commonly known as the *Dangerous Drugs Act*, have put a number of obligations on medical practitioners, which it has taken some time for them to realize. These obligations have been the subject of memoranda issued to members dated October 20 and November 3, 1927, November 29, 1928, February 14 and March 11, 1929. The Act imposes certain duties upon medical practitioners in prescribing the particular drugs; and requires them to keep records of quantities of the drugs obtained by them for use in their practice and the quantities used or remaining in their possession from time to time. It has also put an increased responsibility upon them as being the only persons through whom addicts can obtain their supplies.

British Medical Association Lectures.

In pursuance of the resolution of the Council of January 5, 1926, which established a system of lectures of a distinctive character on scientific and clinical subjects to be delivered at meetings of local associations outside the metropolitan areas, lectures were arranged as follows:

Central Southern Medical Association: (1) Goulburn, August 10, 1928, Dr. R. S. Godsall: Subject connected with ear, nose and throat; (2) Goulburn, February 15, 1929, Dr. C. A. Hogg: "Some Legal Relationships between Psychiatry and the Law."

Northern District Medical Association: (1) Singleton, May 30, 1928, Dr. C. J. Wiley: "Treatment of Gonorrhœa in General Practice"; (2) Armidale, September 19, 1928, Dr. P. L. Hipsley: "Remarks on Certain Deformities, Injuries and Diseases in New-born Children."

Eastern District Medical Association: Kempsey, March 23, 1929, Dr. W. W. Ingram: "Diagnosis and Treatment of Glycosuria and Diabetes from the Point of View of the General Practitioner."

Post-graduate Courses.

A post-graduate work committee as a standing committee of the Council was constituted by the Council at its meeting February 12, 1929. Arrangements in connexion with post-graduate courses hitherto have been a function of the Organization and Science Committee. This branch of the Council's activities will, it is thought, be better developed if in the hands of a committee specially constituted for the purpose.

Arrangements were made with the Melbourne Permanent Committee for Post-Graduate Work for Professor F. R. Fraser, who is Professor of Medicine in the University of London and Director of the Medical Clinic at Saint Bartholomew's Hospital, to lecture in Sydney during his visit to Australia. Professor Fraser delivered two lectures, namely: "The Causes of Dyspnœa and the Clinical Types, with Special Reference to Asthma" on August 28, and "Heart Failure and its Treatment" on August 29, 1928.

Visitor.

Mr. Sampson Handley, an eminent surgeon at the Middlesex Hospital and well known in connexion with research into and the surgical treatment of cancer, made a short call in Melbourne and Sydney on his way to New Zealand. He was appointed the official delegate of the British Medical Association to convey the good wishes of the Association to the Branches he might visit during his journey.

Procedure in Ethical Matters.

In accordance with suggestions received from the Central Ethical Committee of the Council of the British Medical Association, the Federal Committee adopted, April 4, 1928, "Model Rules Governing Procedure in Ethical Matters of a Branch in Australia." These model rules followed the ethical rules adopted by the Representative Body modified to meet the conditions obtaining in Australia. They were received by the Council from the Federal Committee. While they were in process of being redrafted for inclusion in the By-laws of the Branch, the Council was notified by the Federal Committee that the Central Ethical Committee was desirous that they should be further considered by the Federal Committee. The result of this further consideration is now being awaited.

Hospital Policy.

A hospitals committee as a standing committee of the Council was constituted by the Council at its meeting of February 12, 1929. The duties and powers of the Hospitals Committee are to consider and report to the Council upon questions concerning hospitals and medical charities and the professional interests of the medical officers of those institutions.

J. E. V. BARLING,
President.

FINANCIAL STATEMENTS.

DR. W. H. CRAGO, the Honorary Treasurer, moved that the statement of receipts and expenditure be received. The motion was seconded by DR. GEORGE BELL. Dr. Crago also dealt with the balance sheet and financial statement of the premises account. He informed the members that a final settlement had not yet been made by the City Council in regard to the resumption of the house in Elizabeth Street. He gave some details concerning the expenses that had already been incurred in connexion with the new building in Macquarie Street. Of the 1,699 members of the Branch only ninety-eight had taken up debentures. This was a most disappointing response to their appeal and Dr. Crago expressed the hope that those who had not applied for one or more debentures, would do so without further loss of time, as it was eminently desirable that the new building should be financed by money provided by the members. Dr. Crago's motion was adopted.

On the motion of DR. W. H. CRAGO, seconded by DR. J. ADAM DICK, the thanks of the meeting were conveyed to Dr. A. M. Gleddon and Dr. F. W. Hall for their services as auditors.

PRESIDENT'S ADDRESS.

DR. J. E. V. BARLING delivered his address (see page 508).

DR. F. BROWN CRAIG in moving a vote of thanks to the President for his address said that he regarded this as a privilege. Dr. Barling had raised the tone of the Branch above the level of a mere struggle for existence. He had given them a great deal to think about. He had earned the thanks of the members by the way he had carried out his duties as President during the year and especially for the leading part he had taken in steering the Council

BRITISH MEDICAL ASSOCIATION—NEW SOUTH WALES BRANCH.

Receipts and Expenditure for the Year Ended December 31, 1928.

RECEIPTS.	£	s.	d.
January 1, 1928—			
Loan, Premises Account	4,250	0	0
Balance, Petty Cash	6	16	0
Subscriptions	8,121	19	0
Interest	272	10	0
Sales, Agreement Forms	103	7	11
Debit Balance	134	7	0
	£12,888	19	11

EXPENDITURE.	£	s.	d.
Debit Balance as per Cash Book	32	6	11
British Medical Association	2,150	17	9
THE MEDICAL JOURNAL OF AUSTRALIA	2,068	5	0
Clerical Aid	1,682	3	0
Rent, £800, less con. £45	755	0	0
Printing and Stationery	375	1	9
Library	253	3	10
Stamps and Telegrams	190	17	10
Legal Expenses	68	13	0
Travelling Expenses	41	15	1
Typewriter	32	16	0
Electric Light and Power	40	0	11
Sundry Expenses	72	8	10
Telephone	29	2	1
Cleaning Offices	78	0	0
Bank Charges	9	19	7
Loan, Premises Account	5,000	0	0
Balance, Petty Cash	8	8	4
	£12,888	19	11

Examined and found correct.
A. MAITLAND GLEDDEN
FRED. W. HALL
Auditors.
February 28, 1929.

W. H. CRAGO,
Honorary Treasurer.

along a safe course during one or two perilous voyages. Dr. Brown Craig dealt briefly with the work in connexion with the new building and referred to the part taken by Dr. Barling.

The motion was seconded by Dr. C. B. Blackburn and was carried by acclamation.

Election of Office-Bearers.

Dr. J. E. V. Barling announced the result of the election of the President-Elect and members of the Council as follows:

President: Dr. F. Brown Craig.

President-Elect: Dr. E. M. Humphery.

Members of the Council: Dr. George Bell, Dr. W. J. Binns, Dr. C. B. Blackburn, Dr. W. H. Crago, Dr. A. Davidson, Dr. J. Adam Dick, Dr. L. W. Dunlop, Dr. A. J. Gibson, Dr. J. Goodwin Hill, Dr. J. G. Hunter, Dr. C. H. E. Lawes, Dr. T. W. Lipscomb, Dr. R. J. Millard, Dr. A. A. Palmer, Dr. S. A. Smith, Dr. R. H. Todd and Dr. R. B. Wade.

A vote of thanks was accorded to Dr. A. M. Gleddon and Dr. Garnet Halloran, Dr. Cotter Harvey, Dr. V. M. Coppleson and Dr. C. G. McDonald, Dr. M. J. Plomley, Dr. H. A. Ridler and Dr. J. Colvin Storey for their services as scrutineers.

On the motion of Dr. W. H. Crago, seconded by Dr. T. W. Lipscomb, Dr. F. W. Hall and Dr. A. M. Gleddon were elected Honorary Auditors for the ensuing year.

On the motion of Dr. R. J. Millard, seconded by Dr. J. Goodwin Hill, Dr. R. L. Davies was appointed representative of the Branch in the Representative Body, 1929-1930.

On the motion of Dr. W. J. Binns, seconded by Dr. A. J. Gibson, Dr. R. L. Davies and Dr. H. Huff Johnston were appointed delegates of the Branch to attend the annual meeting of the British Medical Association at Manchester.

Induction of President.

Dr. J. E. V. Barling, in vacating the chair in favour of Dr. F. Brown Craig, expressed his thanks to the members of the Council for their unfailing consideration and for their cheerfulness in overlooking his shortcomings. He stated that Dr. Todd, Dr. Crago and Mr. Green had been towers of strength and he eulogized the excellence of the office staff. He stated that there was no need to introduce Dr. Craig who was well known to all the members. He did not think that he was going to have a very easy year, but he felt convinced that he would succeed in carrying out his duties in an admirable manner.

In taking the chair Dr. Brown Craig pointed out that there were two events which would happen during his term of office, to which he looked forward with pleasurable anticipation. They were the holding of the third session of the Australasian Medical Congress (British Medical Association) and the completion of the building in Macquarie Street. He appealed to the members for cooperation to make the congress a great success.

Public Health.

INFLUENZA IN THE AUSTRAL-PACIFIC ZONE, 1928.

(Compiled from reports received through the Epidemiological Intelligence Service, Austral-Pacific Zone, Commonwealth Department of Health, Canberra, Federal Capital Territory.)

Some features of interest are brought out in the reports of influenza in the Pacific during 1928. Complete statistics and records are not yet available, but current reports have been received and transmitted from time to time through the epidemiological intelligence service that now functions as one result of the International Pacific Health Conference that assembled at Melbourne in December, 1926.

It may be said that the latter part of 1928 saw an accession of influenza incidence throughout Australia, in most of the Pacific groups and in California and the middle west of the United States of America. Some features of the reported epidemiological chronology are indicated in the map. There is not shown any definite wave of trans-oceanic spread, but a local spread through island groups must be accepted as fact. One interesting feature is the association of the epidemic at Raratonga with the arrival of the November mailboat from New Zealand. There was not at that time any accession of infection in New Zealand as shown by cases of influenza notified or deaths registered as due to influenza or pneumonia. There had, however, been a definite prevalence of infection in Australia since September and there had been widespread influenza in Samoa, in Niue and in Pukapuka as early as August. It is, therefore, not possible to trace any chronological association along ocean routes. It is, however, of interest to record these Pacific reports since some European epidemiologists have recalled the story of the 1918-1919 pandemic and commented on the rapid development of epidemicity in California during last November and the excessive prevalence of influenza in various parts of the United States that preceded the great pandemic.

The summarized reports recorded are arranged by territory from east to west. The available records from California are also included because of the maritime relationship with the Austral-Pacific Zone which properly embraces the region south of the equator and between longitude 140° east and 140° west.

California.

There was a sharp accession in the number of cases notified in San Francisco and in California during October and November. This is shown in the weekly records of cases and deaths for the State and city from the beginning of September:

Week Ended	California Cases.	San Francisco.	
		Cases.	Deaths.
September 8	24	8	—
" 15	9	4	—
" 22	21	7	—
" 29	23	2	2
October 6	27	9	2
" 13	34	3	4
" 20	171	28	6
" 27	1,557	1,207	10
November 3	2,456	1,114	31
" 10	2,698	716	12
" 17	2,803	992	9
" 24	5,166	327	12
December 1	10,095	214	7
" 8	10,683	137	12
" 15	7,385	88	1
" 22	2,708	56	12
" 29	1,232	41	7

Society Islands.

An epidemic of influenza with numerous cases and some deaths began in the middle of October in Moorea, Makatoa, Isles Sous-le-Vent, Tuomotu and Papeete.

Cook Islands.

In August an outbreak of influenza was reported at Pukapuka, with three hundred cases and three deaths. The epidemic abated in mid-September.

On Saturday, November 17, a week after the arrival of the November mailboat from New Zealand, an outbreak of influenza became evident at Raratonga. There was at this time a succession of cold unpleasant days following on a spell of fine weather and this probably aggravated the condition. The cases were mild and it was estimated by the Chief Medical Officer that some 300 or 400

people must have been affected, chiefly in the settlement of Avarua, with a small number of cases in Arorangi and Titikaveka. By the end of November the epidemic had abated. There have been no deaths attributable to the outbreak. Those who contracted the disease were mostly young adults.

On November 15, two days before the first cases were known in Raratonga, a schooner with cargo and passengers sailed for the Southern Group. It was reported that a similar outbreak make its appearance on board the schooner on the evening of November 16.

Niue.

There was an epidemic of mild influenza reported in Niue in July with three deaths. The epidemic continued during August and abated in September. The total number of deaths was forty. In the three months July-September there were 315 cases of influenza recorded.

Western Samoa.

A wide spread but mild epidemic of influenza in Western Samoa commenced early in July and lasted through August. No deaths were reported.

Tonga.

It would appear that influenza was present in Tonga from April onwards, most prevalent in July and August and thereafter declining. In the three weeks ended April 21 there were 117 cases on the Island of Togatabu and 347 on Vavau. Fortnightly cases on these islands were then reported as follows:

Fortnight Ended	Togatabu.	Vavau.
May 5	52	88
„ 19	29	90
June 2	39	97
„ 16	162	56
„ 30	229	79
July 14	220	66
„ 28	315	144
August 11	244	146
„ 25	119	102
September 8	81	124

The Chief Medical Officer has reported that the attacks were mild in the majority of patients, but in a few there were indications of a *Streptococcus hæmolyticus* infection manifested by hæmorrhages and hæmoglobinuria.

Fiji.

Influenza has been widespread in Fiji throughout the year and towards the end of the year the type was more than usually severe. It is noted that Rotuma which escaped the 1918-1919 epidemic, suffered a severe epidemic in November, 1928, which caused thirty-one deaths in a population of 2,400. It was reported as having been introduced from Fiji. From press reports issued later in February thirty deaths appeared to have occurred from influenza in Futuna, a town with about 1,500 inhabitants and forty deaths in Wallis Island with a population of about 4,500.

British Solomon Islands.

Influenzal colds were prevalent in the British Solomon Islands group particularly during the month of September. There was an increase in the number of pneumonia cases, probably accounted for by this epidemic. The epidemic gradually subsided after September and seemed to be definitely at an end by the New Year.

New Guinea.

There were the usual outbreaks in New Guinea during the third quarter of the year, reports not being received until the epidemic had died down.

New Zealand.

The influenza situation in the Dominion during 1928 presented no features of interest. There was a slight accession of cases of influenza and pneumonia notified in the late winter, but there was no epidemic prevalence indicated. Notifications in four-weekly periods were as follows, the notifications referring to "fulminant, pneumonic or septicæmic influenza" and to "acute primary pneumonia":

Four-Weekly Period.	Influenza. Central Auckland.	Influenza. Central Wellington.	Influenza. Total Dominion.	Pneumonia. Total Dominion.
I	—	1	41	60
II	—	—	11	50
III	3	—	12	43
IV	—	—	5	71
V	4	1	22	60
VI	—	—	22	100
VII	4	1	36	100
VIII	7	—	43	150
IX	5	2	59	178
X	2	2	36	119
XI	1	—	36	151
XII	1	1	37	112
XIII	1	5	23	98

Australia.

The only State in Australia where influenza is notifiable is South Australia. Complete mortality figures are not yet available and it is not therefore possible to indicate the statistical position in regard to the behaviour of influenza in Australia during 1928. It may be said, however, that the earlier winter months saw a usual prevalence of seasonal colds and nothing more. Early August saw influenza becoming prevalent in Sydney. In August and September there would appear to have been a wide spread prevalence through New South Wales and Queensland. Late September and October saw influenza epidemic in Victoria. The type varied from the usual spring influenza of three days' duration to a debilitating illness complicated by a low grade but persistent broncho-pneumonia or an otitis. In mid-November there was reported in Sydney a prevalence of a so-called "one-day" influenza and at the same time some prominence was given in press reports to an outbreak at Leeton, a small country town, 375 miles from Sydney. Within a month forty-two patients were admitted to the local hospital with eight deaths from a complicating broncho-pneumonia. By the end of November with the onset of summer weather the position appeared to be normal in the eastern States. In South Australia, however, it would appear from weekly notifications that a wave of influenza appeared in December and thereafter declined. Up to November 10 the notifications for the State for the year totalled thirty-six. Weekly notifications thereafter were:

Week Ended	Adelaide.	Rest of State.	Total.
November 17	1	—	1
„ 24	13	—	13
December 1	13	—	13
„ 8	57	4	61
„ 15	46	1	47
„ 22	73	18	91
„ 29	13	10	23
January 5	31	8	39
„ 12	2	25	27
„ 19	4	—	4
„ 26	4	5	9

Reports would not indicate that any appreciable mortality was associated with this prevalence of influenza. Quarterly deaths for the year, however, numbered two, none, five and twenty-one respectively. The only complete mortality data on record relate to deaths in the ten Queensland provincial cities and Brisbane, where no accession in the number of deaths attributed to influenza or pneumonia is noticeable.

Corrigendum.

Our attention has been drawn to an error which has been made in the illustrations to Mr. H. B. Devine's article appearing in our issue of April 6, 1929. The illustration labelled Figure XII is the normal cystogram and should be Figure XI. The illustration labelled Figure XI is the cystogram after micturition and shows the growth in the bladder; it is Figure XII.

Books Received.

DIE BEZIEHUNGEN DER PARS TUBERALIS HYPOPHYSIS ZUM HYPOPHYSENAPPARAT, Von Dr. G. R. Cameron; 1929. Jena: Gustav Fischer. Crown 4to., pp. 57.

CATECHISM SERIES: GYNECOLOGY; Fifth Edition; 1929. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 92. Price: 1s. 6d. net.

TUMORS ARISING FROM THE BLOOD-VESSELS OF THE BRAIN: ANGIOMATOUS MALFORMATIONS AND HEMANGIOBLASTOMAS, by Harvey Cushing and Percival Bailey; 1928. London: Baillière, Tindall and Cox. Royal 8vo., pp. 229, with illustrations. Price: 34s. net.

TECHNIQUE AND METHOD OF USE OF STERNBERG'S GASTROSCOPY AND GASTROSCOPIC TREATMENT WITH STERNBERG'S CYSTO-GASTROSCOPIC APPARATUS, by William Sternberg; 1929. London: John Bale, Sons and Danielsson, Limited. Royal 8vo., pp. 19, with illustrations. Price: 3s. 6d. net.

GLEANINGS FROM GENERAL PRACTICE, by David Tindal, M.D., F.R.F.P.S. (Glasgow); 1929. London: Baillière, Tindall and Cox. Crown 8vo., pp. 219. Price: 6s. net.

MEDICAL ADVENTURE: SOME EXPERIENCES OF A GENERAL PRACTITIONER, by Ernest Ward, M.D. (Cambridge), F.R.C.S. (England); 1929. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 291, with illustrations. Price: 8s. 6d. net.

MALARIA PROBLEMS, by Frederick L. Hoffman, LL.D.; 1928. Printed in United States of America by the Prudential Press. Royal 8vo., pp. 207.

Diary for the Month.

APR. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APR. 23.—Illawarra Suburbs Medical Association, New South Wales.
APR. 24.—Victorian Branch, B.M.A.: Council.
APR. 25.—South Australian Branch, B.M.A.: Branch.
MAY 1.—Victorian Branch, B.M.A., Branch.
MAY 1.—Western Australian Branch, B.M.A.: Council.
MAY 2.—New South Wales Branch, B.M.A.: Branch.
MAY 2.—South Australian Branch, B.M.A.: Council.
MAY 3.—Queensland Branch, B.M.A.: Branch.
MAY 7.—Tasmanian Branch, B.M.A.: Council.
MAY 7.—Eye, Ear, Nose and Throat Section, South Australian Branch, B.M.A.
MAY 8.—Central Northern Medical Association, New South Wales.
MAY 9.—Victorian Branch, B.M.A.: Council.
MAY 9.—Section of Orthopædics, New South Wales Branch, B.M.A.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

BALMAIN AND DISTRICT HOSPITAL: Honorary Pathologist.
CHILDREN'S HOSPITAL, CARLTON: Honorary Medical Officer.
CITY OF BENDIGO: Medical Officer of Health.
DEPARTMENT OF DEFENCE, ROYAL AUSTRALIAN NAVY: Surgeon Lieutenant.
DEPARTMENT OF INSPECTOR-GENERAL OF HOSPITALS, "MAREEBA" BABIES' HOSPITAL: Honorary Aural Surgeon, House Surgeon.
INSPECTOR-GENERAL OF HOSPITALS' DEPARTMENT, ADELAIDE HOSPITAL: Honorary Deep X Ray Therapist.
THE BRISBANE AND SOUTH COAST HOSPITALS BOARD: Honorary Urologist.
THE UNIVERSITY OF MELBOURNE: Chair of Obstetrics.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital. Boonah District Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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